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Medical Care of the Veteran

PAUL B. MAGNUSON*

VETERANS ADMINISTRATION, WASHINGTON, D.C.

During the past few years many of my friends throughout the country have accused me of being a bureaucrat because of my association with the Federal Government, or more specifically, with the Veterans Administration. I want to take this opportunity to demonstrate to you that we in the Veterans Administration are not attempting to practice bureaucratic medicine, but that on the contrary our plans call for the practice of medicine on the level of the local communities in which the VA installations are located.

I first entered the Veterans Administration picture in 1942. At that time we had over 10,000,000 men in the armed forces and it seemed to me there was a need for planning for the expansion of veterans hospitals. With that thought in mind I prepared a plan for the administration of veterans hospitals which I believed would overcome most of the objections to the operation of such hospitals in the past, and I journeyed to Washington to discuss my plan with General Hines, who was then the Administrator for Veterans' Affairs.

Nothing further happened until 1945, when General Bradley became Administrator of Veterans' Affairs and brought with him as his Chief Medical Director General Paul R. Hawley. Shortly after they entered on the job General Bradley and General Hawley called on me and discussed the ramifications of my plan, with the result that I, too, went to Washington with a promise from General Bradley that I would have a free hand in implementing the plan. Incidentally, when I agreed to come to Washington I asked my school, Northwestern University, for six months' leave, intending to stay only that long, which shows the exaggerated ego some of us have when we take on a new job.

On my arrival in Washington I learned that professional personnel in the Department of Medicine and Surgery were rated under Civil Service classification standards and all men in the department had to come off Civil Service rolls. This was, in my opinion, an obstacle to the proper implementation of the program as we planned it. Fortunately, Generals Bradley and Hawley had already recognized that medical men could not work successfully, if hampered by too many regulations, and had had a bill introduced into Congress which subsequently became Public Law 293, which removed doctors, dentists, and nurses in the Veterans Administration from Civil Service jurisdiction and placed them under a special professional personnel system under the control of the Department of Medicine and Surgery. This bill was signed by the President early in 1946, and we proceeded to install our plans, which in effect are as follows:

We in Washington make no attempt to practice medicine in any part of the country, not even in Washington. Instead, we ask the doctors in each local community to participate in the treatment of patients who are charged to the Veterans Administration by law. We appoint full-time medical men in our teaching hospitals only on the recommendation of the Deans Committees, which are committees consisting of faculty members of medical schools in the communities served by our VA hospitals. As a result our staff in these hospitals consists of top-flight men, including many professors from medical schools who are now full-time chiefs of service in veterans hospitals. Throughout the country the medical schools have taken hold and we now depend on them for recommendations for the selection and appointment of residents, attending physicians, and consultants.

In most of the outlying hospitals which are not connected with medical schools we have consultants visiting at least once a week to make ward rounds, examine patients, and give seminars and lectures to which the medical profession in the community is invited.

In addition, in hospitals in communities where there are no medical schools, we use men in the

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Presented November 9, 1948, before the meeting of the Interstate Postgraduate Medical Association of North America, Cleveland, Ohio.

PAUL B. MAGNUSON

community for staffing on a consultant and attending basis. We cannot have residents in all of these hospitals because there are not enough specialists available to conduct firm residency training programs, but we do have consultants going there constantly to give instruction. We also have a system whereby any hospital can invite any specialist, from anywhere within the continental limits of the United States to come and examine patients, lecture, and give seminars. We in Washington do not tell them whom to have; all we do is give them the money to pay these specialists.

We are making an effort to put the care of veterans, with which the Veterans Administration is charged by law, in the hands of the local medical men, and to pay them for it. Our hospitals run, as nearly as we can run them, on the same principle as university or teaching hospitals. We will help to spread the medical knowledge that we gain constantly to the men who are busy practitioners and do not otherwise have the time to keep in touch with the latest developments in medicine.

Our medical program also includes the outpatient clinics at regional offices throughout the country. These clinics are established for treatment of service-connected conditions only, on an outpatient basis. At the present time, we are not completely satisfied with the condition of these clinics, but we are constantly endeavoring day by day to improve the quality of treatment.

Somebody says, "What about the nonservice-connected disabilities? What about the fellows who can afford to pay a fee but who are going into veterans hospitals?" Let me tell you how the law reads, then it is up to you to correct that. The laws says that if there is a vacant bed in a veterans hospital, a nonservice-connected veteran can demand that he be taken into the hospital. Most of them are medically indigent. They have to sign what we call a Form P-10, which veterans organizations call a "Pauper's Oath," in order to get this medical service. That P-10 provides for a thousand dollar fine if it is sworn to falsely, but it is also provided in the law that the Veterans Administration cannot go behind the statement of the veteran and attempt to prove whether or not it is true.

We naturally feel that people should not impose on this sort of service, but I suppose the better the service gets the more we will be imposed upon. There is some abuse of the privileges granted by this law, but the number of cases is actually very small. At any rate, we in the Veterans Administration are powerless under the law. We cannot change the law. If it is to be changed it is up to somebody else.

We now have 126 hospitals accommodating over 108,000 patients. In addition, Congress has authorized the construction of more hospitals, which when completed will give us a total of approximately 141,000 beds. It is said that we cannot possibly staff properly for more than 120,000 beds, and so we will probably be accused again of practicing or trying to extend the practice of bureaucratic medicine. I have tried to show you this evening that such is not our intention.

We in the Veterans Administration are attempting to administer the laws passed by Congress to the best of our ability. If you, as members of the medical profession, object to some phases of our program you can help change them by using your influence with your neighbors. They are the taxpayers and the voters, and as we observed on November 2, the final determination on the policies of the Government of this country rests with them.

Polypoid Disease of the Colon

LOUIS A. BUIE*

MAYO CLINIC, ROCHESTER, MINNESOTA

Probably 12 to 14 per cent of persons more than 40 years of age have polyps in the colon. One of every 10 persons of this age group has a polyp which can be discovered on proctoscopic examination. Recently 910 patients, who made no complaint of abnormal intestinal habits, were examined proctoscopically at the Mayo Clinic and, in the terminal portions of the colons of 76 of them, polyps were discovered. This incidence of almost 10 per cent does not differ materially from that obtained from examination of patients who complain of abnormal intestinal habits.

All polyps situated within reach of the proctoscope should be destroyed forthwith and, as will be shown, most of them can be destroyed by fulguration. This is not to imply that polyps which occur higher in the colon should be considered benign. They, also, should be destroyed but their destruction falls in the field of general surgery.

Approximately 30 per cent of colonic polyps are situated in portions of the colon which cannot be inspected on proctoscopic examination. One may well devote thought to methods of detecting these growths, many of which are so small that they cannot be discovered on roentgenologic examination.

All polyps of the colon are cancers (carcinomas) or will become cancers if they are not destroyed and if the patient lives long enough. This extreme belief is a safe one to hold and there is much evidence, not always controlled of course, to support it. For example, Brust and I[1] studied the records of 200 patients who had polyps which could be seen through the proctoscope. All of these patients were advised to permit destruction of the polyps by fulguration. Four of the patients refused to accept this advice. All 4 returned within three years complaining of intestinal symptoms and, in examination of each, a cancer was found at the site of the polyp which had been discovered by us. (Figure 1.) Further evidence of the probable significance of the relationship between polyps and cancers is the fact that 25 to 30 per cent of all patients who have cancers of the colon have associated polyps and usually the polyps are situated in the tissues adjacent to the malignant growths. It is known, also, that most polyps possess adenomatous structure and many of them undergo various degrees of malignant change. I believe, as I said at the outset of this paragraph, that all polyps of the colon would develop malignant characteristics should their hosts live long enough.

Therefore, the most important question concerning polypoid disease of the colon is not *whether* something should be done about the lesions but *what* should be done.

We physicians know how the disease manifests itself and we know what symptoms it produces. We know the distribution by age and sex and the comparative frequency with which polyps involve the various segments of the colon. We understand

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Presented November 10, 1948, before the meeting of the Interstate Postgraduate Medical Association of North America, Cleveland, Ohio.

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the morphologic characteristics of the growths in all types of polypoid disease and we are aware of the tendency of certain types of polyposis to "run in families." Nevertheless, despite the fact that this condition is so accurately known, methods of therapeutic attack are limited and, when applied, often are attended with discouragement.

The morphologic characteristics of the growths in polypoid disease vary widely but the distribution of polyps of the colon shows certain definite tendencies. Consequently, several significant factors must be considered when an attempt is made to determine the method of treatment which should be utilized in any particular case. I shall explain these factors and shall discuss the type of treatment which I believe is indicated.

Half of the polyps of the colon are small, sessile excrescences, 2 to 8 mm. in diameter. Most of these small, tuft-like lesions are discovered in the course of routine proctoscopic examination and, as a rule, they bear no relationship to the chief complaint of the patient. In fact, owing to the minuteness of the lesions, practically none of these patients is aware of disability referable to the bowel. Many are small mammillations which cast doubt concerning their significance in the mind of the physician, but he should never disregard them. Large, sessile polyps are even more important. (Figure 2.)

LARGE, SESSILE POLYPS

A sessile polyp, 15 mm. in diameter or larger, requires particularly serious consideration. Polyps may attain enormous proportions and fill the rectal ampulla or other segments of the colon. They may grow from a small portion of the bowel wall or the mucous membrane may become occupied by a polypoid mass which leaves little or no normal tissue within the diseased segment. (Figure 3.)

Repeated microscopic examination of polyps of all classifications may disclose no malignant change but, just as often, such transition will be discoverable. Therapeutic measures which should be applied in the presence of such massive involvement of the colon are essentially the same as those which are employed in connection with malignant colonic disease. All effort should be directed toward eradication of the tumor and of the segment of colon which is involved. Complete destruction of the lesion, of the portion of bowel wall to which it is attached, and of the tissues adjacent to the bowel wall may be possible by fulguration and should be given proper consideration. By this method, often, the disease can be eradicated, continuity of the bowel can be maintained, and performance of colostomy may be avoided.

At this point I should emphasize the significance and the importance of the physician's attitude when colostomy is being discussed with his patient. Patients have a horror of colostomy and they cannot be blamed for that, but I have profound lack of regard for a physician who will join with his patient and express a sentiment which may carry just enough weight to influence the patient against acceptance of a surgical operation which might save his life. It is the duty of the physician to explain to his patient that the colonic stoma can be cared for with little effort and that, by the use of proper dressings and abdominal supports, little inconvenience is experienced. The patient should know that these factors are insignificant when compared with the major considerations.

As I have implied, if a solitary lesion is situated in a portion of the bowel which can be reached through the proctoscope, if it is not too large, and

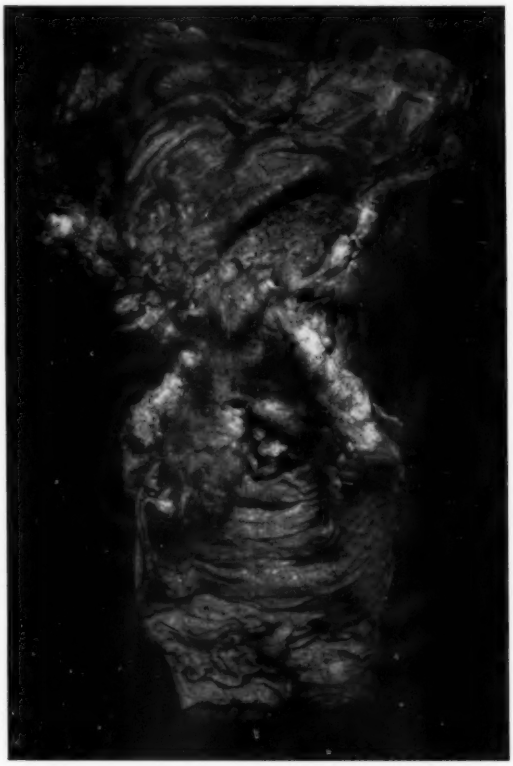

Figure 1. Resected specimen of adenocarcinoma of the rectum. The peculiar alteration in the mucosa adjacent to the lesion and the sentinel polyp near the bottom of the specimen can be seen.

Figure 2. A small, single polyp.

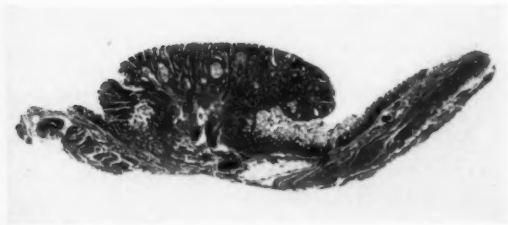

Figure 3. Sessile polyp.

if destruction of the lesion along with that portion of the bowel wall to which it is attached will not injure vital structures adjacent to the bowel, fulguration can be employed. If such a lesion is discovered in a portion of the colon which lies within the peritoneal cavity, or which is adjacent to such structures as the urinary bladder, the uterus, the vagina, the prostate gland and other pelvic structures, then laparotomy should be performed and the tumor should be resected.

The type of operation which should be employed depends on the position, size, and morphologic characteristics of the growth. If it is attached to the bowel wall by a pedicle, incision of the colon, followed by ligation and excision of the polyp near the base of the pedicle, and immediate closure, will suffice. If the bowel wall is involved in the pathologic process, often it is necessary to take away that portion of the bowel which is diseased, in much the same manner as that employed when dealing with carcinoma.

PEDUNCULATED POLYPS

Some polyps are attached to the wall of the colon by stems of mucous membrane and muscularis mucosae. Sometimes the serosal surface of the bowel is drawn into this pedicle. The diameter of the body of the polyp varies from a few millimeters to that of a mass which may fill the lumen of the bowel. The length and diameter of the pedicle vary widely. These polyps may occur singly or in scattered collections. Most single polyps of this type can be eradicated by fulguration and it is advisable to use this method instead of applying a loop cautery. The latter may cut into the sub-

Figure 4. Mounted specimen of large pedunculated polyp. Such a tumor will slough away following fulguration of its stem.

stance of the polyp or the pedicle and cause dangerous hemorrhage. (Figures 4 and 5.)

The view has been advanced that the cautery should be employed so that a specimen can be obtained for microscopic examination. Such a specimen can be obtained with a biopsy forceps prior to fulguration but the factor of safety would prejudice most competent physicians in favor of fulguration. Furthermore, I doubt that the discovery that a polyp is benign would alter the treatment.

Figure 5. A large polyp. Such a tumor in the sigmoid may produce symptoms because of hyperactivity due to its presence. The muscular layer is drawn into the pedicle. Application of a snare to such a pedicle might result disastrously.

DIFFUSE POLYPOSIS

In this condition, little or no normal wall is left within the segment of colon which is involved. "Multiple polyposis," "disseminated polyposis" and "polypoidosis" are terms used to designate the condition. Total involvement of the bowel wall is one of the distinguishing characteristics of the disease but, in addition, the wall itself may be altered by diffuse hyperplasia of the mucous membrane. Sometimes polyps with pedicles, sessile polyps and diffuse hyperplasia of the mucous membrane are associated in this disease and often the condition is part of a form of polyposis in which the wall of the entire colon is occupied by polyps. (Figure 6.)

For treatment of this condition, fulguration should be employed only when other types of treatment are contraindicated; for example, radical surgical operation may be contraindicated because of some systemic disorder. Fortunately, massive or disseminated involvement of the bowel is seen rarely. In a recent review of a large series of records of patients with polypoid disease of all types, I found that there were only two instances of disseminated polyposis in which it was felt that fulguration should be adopted.

"MULTIPLE POLYPOSIS"

In this category are included, at one extreme, patients whose condition may be totally unproductive of signs or symptoms and, at the other, the individuals whose colons are completely involved by diffuse hyperplasia and polyps. This is the heredofamilial type of the disease and is the condition which is known as "multiple polyposis." Colectomy is the only successful treatment for this condition. However, by removing that part of the polyp-bearing colon which is not accessible through the proctoscope, and then applying fulguration to that portion which is thus accessible, the resection can be reduced to partial colectomy. (Figure 7.)

Figure 6. Multiple discrete polyps. One polyp is large and malignant.

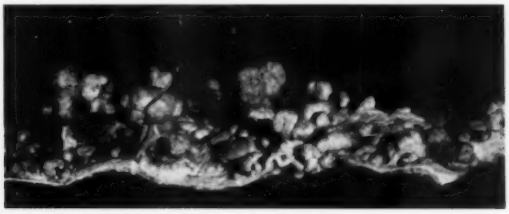

Figure 7. Multiple polyps with diffuse hyperplasia of the colonic wall.

Owing to the extreme risk of partial colectomy, it is advisable that this operation be performed, as has been indicated, before fulguration is begun. I prefer this arrangement because I have seen patients succumb following colectomy which had been performed after they had spent endless days undergoing removal of polyps from the rectum and the sigmoid by fulguration. Sometimes many months are required to destroy all the growths in the rectum and sigmoid. Often several hundred of these lesions must be fulgurated and often it is necessary to allow several weeks to elapse between treatments. It is deplorable if, following such a trying experience, the patient comes to colectomy and fails to survive.

After partial colectomy, the rectum and the lower portion of the sigmoid can be cleared of polyps by fulguration and, later, ileosigmoidostomy can be performed. At the first operation a temporary ileac stoma can be created and the proximal end of the stump of the sigmoid also can be brought out as a stoma in the left lower portion of the abdominal wall. Campbell[2] has described a procedure wherein he brings part of the ileum and part of the sigmoid out through "the lower angle of the incision so that a spur clamp [can] be applied and closure effected later." Thus fulguration of the high polyps can be carried out through the abdominal stoma, as well as through the rectum.

THERE are two morphologic types of multiple polypoid diseases of the colon. In one, the polyps are discrete and the wall of the bowel between them is relatively little involved. In this type, accessible polyps can be fulgurated and, when the bowel has healed, ileosigmoidostomy can be performed. Much has been accomplished by this method and much more may be accomplished. In the other type, polypoid tumors are superimposed on diffusely hyperplastic mucous membrane and there is no possibility of applying fulguration for the polypoid disease because such a procedure would destroy the bowel itself. If fulguration is attempted, there is left only a scarred, contracted remnant, of which the lumen will not be sufficient to permit satisfactory or comfortable passage of feces. Such a strictured tube is poor material for anastomosis with the ileum.

Roentgen therapy apparently has no curative effect on polypoid disease of the colon. Occasionally a physician has felt that beneficial effects have been produced in isolated cases but nothing more. All that is justifiable at present is hope for the future.

Figure 8. Fulguration of high rectal or sigmoidal polyp; *a*, suction pipe to exhaust smoke; *b*, light carrier; *c*, polyp; *d*, applicator.

These illustrations are reprinted with permission from: Buie, L. A.: Practical Proctology. Philadelphia, W. B. Saunders Company, 1937, 512 pp.

TECHNIC OF FULGURATION AND SELECTION OF PATIENTS

For fulguration, a monopolar current is employed. The object of fulguration is complete destruction of the growth by fire. The tip of the electrode can be placed directly against the surface of the tumor. This dries out the tissues. The field to which fulguration is applied must be kept dry. After a field has been dried out in this manner, the tip of the applicator is removed from contact with the lesion and is held at a point approximately 6 or 8 mm. away from the tissues, so that the spark will leap from the electrode to the surface of the growth. An actual flame will develop between the tip of the electrode and the tissues which are being destroyed. The amount of current required in any given case is that which is sufficient to create the tongue of flame between the tip of the electrode and the surface of the tumor. This factor varies not only with patients but with different electrical machines and is best determined by personal experience.

A proctoscope which is suitable for use while performing fulguration includes a built-in suction tube through which smoke and gas can be exhausted from the bowel during the operation. If such suction is not available, gas within the bowel may ignite or even explode. Also, means must be provided for exhausting fecal matter and fluids from the bowel. (Figure 8.)

In most instances, anesthesia is not necessary or desirable. General anesthesia never should be employed. When the lesion is close to, or in, the anus, or when adequate exposure cannot be obtained, caudosacral or low spinal anesthesia can be used.

It is well to consider here, again, that although fulguration is an effective method great care should be exercised when selecting patients for this type of treatment. Sometimes a lesion may possess characteristics which are favorable for fulguration, yet to employ it may be unwise or even hazardous because of other unfavorable characteristics.

Small, sessile lesions can be fulgurated with little regard to their position in the bowel. Large, sessile polyps require more careful consideration. If such a polyp must be fulgurated, the ideal situation for it is on the posterior wall of that portion of the rectum which is not covered by mesentery. In that location there is little danger of injuring any of the vital structures.

Usually an accessible pedunculated polyp can be fulgurated, regardless of its size or position, provided adequate exposure of the polyp and of the entire pedicle can be obtained. The method of applying fulguration to such a polyp is important. The attack should not be made on the pedicle, with the purpose of causing the free-lying portion of the polyp to slough off quickly, in the hope of shortening the duration of the procedure and of the convalescence. It is better to proceed with caution and to fulgurate the body of the polyp, even if fulguration cannot be completed at one time. In

this manner, the polyp will disintegrate, slough away slowly and probably will not leave an open vessel in the stump of the pedicle. Thus, the danger of hemorrhage is reduced.

Large, disseminated, polypoid lesions and those which are associated with diffuse hyperplasia of the mucous membrane respond poorly to fulguration. Radical surgical treatment is indicated in such cases as these.

OBSERVATION AFTER TREATMENT

Regardless of the type of treatment employed, the physician is never justified in releasing the patient entirely from observation. This is particularly true in those cases wherein fulguration has been employed. In a few cases in which a single adenoma, or several adenomas, of the rectum or lower portion of the sigmoid have been fulgurated, the lesion has recurred. Moreover, I have observed that in 21 per cent of cases in which these lesions have been destroyed, other polyps have been discovered later in tissues adjacent to the site of the destroyed polyps. This confirms the opinion that some individuals have a tendency to grow polyps and, once a polyp has been discovered in a colon, its host should be examined periodically thereafter, whether symptoms are present or absent.

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WOMEN PHYSICIANS HONORED

The one-hundredth anniversary of women's entry into the medical profession was observed this year with ceremonies at which 17 contemporary women physicians of the United States, Canada, England, and France were honored for outstanding contributions in their fields. The date marking this century of progress was the anniversary of the graduation of Dr. Elizabeth Blackwell, first woman in the world to receive a medical degree.

At the New York Infirmary, New York City, Elizabeth Blackwell citations were presented by Mrs. Frank A. Vanderlip, president of the board of trustees, to five New York women. In a ceremony at Hobart and William Smith Colleges, Geneva, New York, Mrs. Eleanor Roosevelt announced the names of 12 chosen for honors from the international field.

Those honored at the New York City observance were Dr. Lauretta Dender, for achievements in child psychology; Dr. Connie Myers Guion, internal medicine; Dr. Anna Hubert, surgery; Dr. May Wilson, pediatric cardiology, and Dr. Ada Chree Reid, cardiology.

Cited at the Geneva ceremony were Dr. Priscilla White, Boston, for work in diabetes; Dr. Florence R. Sabin, Denver, medical research; Dr. Alice Hamilton, Hadlyme, Connecticut, industrial medicine; Dr. Helen V. McLean, Chicago, psychiatry; Dr. Helen B. Taussig, Baltimore, clinical medical research; Dr. Margaret D. Craighill, Topeka, Kansas, military medicine; Dr. Martha May Eliot, Washington, public health; Dr. Gerty T. Cori, Webster Groves, Missouri, biochemistry and pharmacology; Dr. Helen MacMurchy, Toronto, child welfare and public health; Dr. Elise S. L'Esperance, New York, pathology and cancer prevention; Dr. Helen M. M. Mackay, London, pediatrics, and Dr. Therese Bertrand Fontaine, Paris, first French woman medecin des hopitaux.

UNIVERSITY OF ILLINOIS INSTALLS BETATRON

A 22-million volt betatron for use in cancer research and treatment has been installed at the University of Illinois College of Medicine, Chicago, which will pioneer in the medical use of this instrument. Tests by Professor Donald W. Kerst of the University's physics department prove that the high energy betatron has the medical advantage of concentrating more effect inside the subject than the x-rays now in use for treating deep cancers.

Emergency Treatment of Apoplexy

GEZA DE TAKATS*

UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE, CHICAGO

THE attitude of the medical profession toward strokes has become one of nihilism. This situation has arisen for several reasons: (1) There has not been a systematic effort to separate the three major causes, namely embolism, thrombosis, and hemorrhage, from each other, although this can be done in the majority of instances. (2) The cerebral damage has been thought to be permanent, irreversible, and unalterable. (3) Some patients have made such a good recovery without specific treatment except good nursing care, that any therapeutic attempt has been regarded with suspicion and thought to be superfluous and not responsible for the spontaneous improvement.

It is difficult to estimate the number of strokes occurring day after day in this country but the tremendous amount of residual damage following apoplexy is obvious to everyone. At St. Luke's Hospital, Dr. Gilbert and I studied 121 patients with apoplexy between 1940 and 1946 and found 15 cerebral emboli, 53 cerebral thromboses, and 53 cerebral hemorrhages. Table 1 indicates our diagnostic criteria based on this material.

CASE REPORT[1]

A 37-year-old white woman was admitted to the Cleveland Clinic Hospital on September 19, 1947, in a comatose state. According to the history obtained from the mother, she had been treated for high blood pressure for a year previously, but she never complained of headaches or visual disturbances. She was found unconscious at work on September 17, 1947, and apparently had vomited.

Lumbar puncture at a local hospital on September 18 revealed many red and white blood cells. There was a paralysis of the right arm and leg. Urinalysis revealed a 4+ albuminuria; blood pressure was 220/110, pulse 92, respiration 24. The pupils were small, round, and equal, and reacted to light. Fundus examination revealed 3+ constriction and 2+ sclerosis of retinal vessels. A systolic precordial murmur was present. There was right facial weakness, paralysis of the right arm and leg, right hyperreflexia, and right Babinski. Lumbar puncture: initial pressure 220 mm. of water; 12 cc. pink cerebrospinal fluid removed; final pressure 110 mm. of water. The patient was conscious by September 20, but when she was discharged on September 24, she still had complete aphasia, right hemianopsia, and right hemiplegia.

She was readmitted approximately three weeks later because of increasing helplessness, and increased difficulty in eating; she kept her head turned to the left. All the physical findings present on the previous admission were still noted, in addition to bilateral papilledema with retinal hemorrhages and exudates, nuchal rigidity, and crepitant râles at the left base. Lumbar puncture again revealed xanthochromic fluid and a high pressure. The initial pressure at the previous admission was 220; at this time, it was 190.

An encephalogram made at this time was not diagnostic; she had right stellate block on October 11, 1947, and it was not followed by any improvement.

I would like to point out that she still shows considerable spasticity on the right hemiplegic extremity, so much so that she requires a brace to enable her to walk. She is regaining her speech gradually. Her improvement has been rather slow, and it is my opinion that she had a cerebral hemorrhage.

Cerebral embolism was diagnosed in patients with sudden cerebrovascular insult, who had an

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Presented November 12, 1948, before the meeting of the Interstate Postgraduate Medical Association of North America, Cleveland, Ohio.

[1]This patient was presented to the Assembly through the courtesy of the local committee. She belongs to the group of cases in whom sympathetic block need not be done.

obvious cardiac lesion for the source of the embolus, or who had had previous embolic episodes to the brain or elsewhere in the body, whose spinal fluid was clear and showed no elevation of pressure. The ages in this series of 15 patients varied from 18 to 60, the average being 53 years. The blood pressure varied from 110/80 to 210/100. Seven patients belonged to the hypertensive arteriosclerotic group and 8 to the rheumatic group. Of the 6 patients with cerebral emboli based on rheumatic heart disease only 1 died, a 40-year-old male, with heart failure; of the remaining 9 patients having cerebral emboli as postcoronary complications and hypertensive cardiovascular disease, 5 died. While this is of no statistical significance in such a small series, it is obvious that the prognosis is better in the rheumatic group.

Cerebral thrombosis, or more appropriately cerebral softening, shows areas of malacia or necrosis in the brain as a result of stenosis or hyalinization of arterioles, whereas an arterial thrombosis is seldom encountered. In this group of 53 patients, 9 died in the attack. A fall in blood pressure and the anoxia of the inevitable bronchopneumonia of the unconscious patient seemed to transform the silent or latent cerebral softening to the clinical entity of "thrombosis" or even to death.

To make a diagnosis of encephalomalacia—not embolus and not hemorrhage—we postulate a patient afflicted with cardiovascular-renal disease, moderate hypertension, and an onset which is frequently gradual. While loss of consciousness was present in approximately one-half of the cases, it did not persist for many days. Increased intracranial pressure was not observed, the spinal fluid was clear, and if the pressure of the spinal fluid was elevated, it was part of the hypertensive encephalopathy. This group is really diagnosed by exclusion of embolism and hemorrhage from the causes of stroke.

The average age of this group was 60.8 years. The average blood pressure need not be stressed since the study of the charts revealed that readings were determined at various stages of the attack and therefore are meaningless. It is an interesting fact, however, that postoperative or postcoronary hemiplegias were noted to be accompanied by a prolonged and rapid fall in blood pressure. This was also true in some strokes occurring during sleep. The onset was sudden in 25 and gradual in 28 of the patients.

If the patient did not die of the attack or of complicating bronchopneumonia, recovery was slow, improvement being noted in 20 and no improvement in another 20 patients. Fatalities were recorded in 13 patients (24.5 per cent) all of them dying between the fifth and twenty-eighth day after the accident.

GEZA DE TAKATS

Cerebral hemorrhage was diagnosed in 53 patients; of these, 41 died within a few hours or days, a mortality of 77 per cent. The average age of this group was 58.9 years, ranging from 41 to 80 years. The average blood pressure was 213/120 with extremes of 300/150 to 120/70. Again it should be pointed out that the vascular insult itself may modify the blood pressure readings but the tendency for the highest pressures among the three varieties of apoplexy is well defined in cases of hemorrhage. Only a few patients showed a gradual onset, but even in these a deepening coma was observed. A sudden, fulminating onset was observed in 49, or 92 per cent, of the 53 patients. The spinal fluid was clear in 30 per cent. Over two-thirds of the patients had bloody spinal fluid and three-fourths had bloody spinal fluid or increased spinal fluid pressure (over 160 mm. of

TABLE 1

SUMMARY OF FINDINGS IN 121 CASES OF APOPLEXY

	DIAGNOSIS		
	EMBOLUS	HEMORRHAGE	THROMBOSIS (*Cerebral softening*)
Number of patients	15	53	53
Average blood pressure	143/85	213/120	166/97
Sudden onset, per cent	100	92	47
Normal spinal fluid, per cent	100	30	90*
Improvement noted, per cent	40	9	38
Mortality, per cent	40	77	24

*No blood, but increased pressure due to hypertension.

water). In massive hemorrhage the outlook was poor, only 9 per cent improving; 14 per cent were unimproved, and 77 per cent died (Table 1).

Attention should be called to the fact that in nonfatal hemorrhage the spinal fluid may be free of blood in one-third of the cases, and the pressure is not elevated in one-fourth of the cases. On the other hand in patients with cerebral softening, the spinal fluid pressure was increased in 10 per cent of the cases, due to hypertension. A second spinal tap done four to five days after the original insult may reveal red cells, frank blood, or increased pressure in patients with a slow leak of their intracerebral hemorrhage in the ventricular system or to the surface of the brain.

COURSE OF EVENTS IN THE BRAIN

Our knowledge regarding the vascular phenomena in the cranial cavity following a stroke is based on animal experiments studied through a transparent window in the skull, on occasional operative findings in man, and on a huge mass of postmortem observations. The most clearcut evidence of a vasomotor storm is found after an experimental embolus, when the vessels of the pia are seen to contract around the embolus. Interestingly this does not occur in air-embolism, but only in thrombo-embolism. Small cerebral emboli are often seen in fibrillators suffering from rheumatic heart disease or in patients with intraventricular thrombi following myocardial infarction.

Patients with so-called cerebral thrombosis really seem to have a gradual narrowing and sclerosis of their cerebral vessels; the ischemic areas of the brain undergo softening. The areas of malacia get a slight blood supply through the main channel and some collateral channels, which exist in the brain just as elsewhere. When blood pressure falls during sleep, in shock after a coronary occlusion, after a pulmonary embolus, or during a surgical operation, the partial anoxia becomes total, and signs and symptoms of a cerebrovascular accident occur. In this softened area hemorrhage may occur through diapedesis.

In hemorrhage, rupture of a normal vessel can hardly occur with blood pressures of 300 millimeters of mercury. It is necessary that the wall be weakened by hyalinization, aneurysm formation, or such increase in fragility that sudden fluctuations of pressure produce petechiae or coalescent hemorrhages. The Rumpel-Leede test for capillary fragility is surprisingly often positive in late stages of hypertension, in avitaminosis, in allergies, in chronic alcoholism, and many other states.

The diabetic retinopathy with hemorrhages seems to be favorably influenced by rutin (the permeability vitamin) and hence its administration to patients suffering from cerebral hemorrhage may have preventive value. Since 20 per cent of 1,200 hypertensives showed increased capillary fragility, at least this group should get rutin for prophylaxis of cerebral hemorrhage.

THERE may be, of course, other reasons for acute cerebral anoxia, such as a long-lasting nitrous oxide anesthesia with insufficient oxygen supply, or a prolonged operation on an exsanguinated individual, on a blue baby, or hypertensives used to higher pressure. With the advent of the long-lasting operations, with or without profound loss of blood, the involvement in cerebral circulation must be considered.

From the standpoint of localization it should be remembered that the majority of vascular accidents occur in the region of the internal capsule, from where the motor, the sensory, and visual phenomena originate. The lesion in the basal ganglia affects the extrapyramidal system and thus is responsible for the rigidity of the hemiplegic limb. The cortex itself may only be involved in collateral edema and produce flaccidity. Flaccidity followed by spasticity means the disappearance of cerebral shock and is the expression of the actual organic damage. We shall see that in our therapeutic attempts this phenomenon is of significance.

The internal capsule is supplied by the perfor-

ating branches of the middle cerebral artery, which have been compared to the bristles of a beard. These are the vessels most frequently involved in a cerebrovascular accident and, depending on the extent of hemorrhage or softening, there will be contralateral hemiplegia, sensory changes, facial changes, and sometimes ipsilateral involvement. It is well for all of us treating cerebral vascular lesions to have at least a rough conception of the area involved.

TABLE 2

APOPLEXIES—CRITERIA OF IMPROVEMENT FOLLOWING STELLATE BLOCK

	NUMBER OF PATIENTS
Regained consciousness	6
Regained speech	5
Improved speech	12
Motor improvement	8
Abolished flaccid paralysis	10
No improvement	9

OUTLINE OF TREATMENT

THE earlier treatment is begun the better are the chances for hastening restitution. A brief cardiovascular and neurologic examination is done and the history is obtained from the relatives. The patient is immediately placed in an oxygen tent; this not only helps his anoxia but may indirectly aid his aortic depressor and carotid sinus reflexes. If signs of increased cerebrospinal pressure are dominant, a spinal tap is done and spinal pressure is measured. Frank blood indicates cerebral or subarachnoid hemorrhage. Marked hypertension with signs of increased venous pressure in the neck requires venesection, which is done slowly, not more than 300 cc. of blood being removed at one time. Unless coma is deepening and a terminal condition is obvious, and if massive hemorrhage can be reasonably excluded, a sympathetic block is performed with the patient in the reclining or semisitting position in his bed.

For diagnostic and therapeutic purposes we have generally preferred the anterior approach to the sympathetic chain. The method used is as follows: The patient's neck is slightly hyperextended by placing a small pillow under the shoulder blade of the same side; the head is turned away from the site of injection. With an applicator dipped in iodine, a line is drawn from the mastoid process to the clavicle, through the tips of the palpable transverse processes. A dermal wheal with 1 per cent procaine (without epinephrine) is placed over the tip of the seventh transverse process. A 4-inch (9 cm.) 22-gauge needle is inserted through this wheal. It must shortly make contact with the tip of the transverse process, which is quite superficial. Then the needle slides along the superior border of the transverse process until it contacts the body of the sixth cervical vertebra. Aspiration is now made for air bubbles, for blood, and for spinal fluid; if this is negative, 10 cc. of 1 per cent procaine solution are injected, aspirating repeatedly during this procedure.

A successful block must be followed within ten to fifteen minutes by a Horner's syndrome, by a dilatation of the conjunctival vessels, by a dryness and increased warmth of the face and ear lobes on the injected side, and by a rise in temperature and dryness of the corresponding upper extremity. Unless a Horner's syndrome develops, the injection must be regarded as faulty and should be repeated.

This cervical injection is done on the side of the lesion; this is not immediately obvious when the patient is seen quite early, since he may be quadriplegic and have a flaccid paralysis, though either a facial involvement or a beginning sensory change may point to the side of involvement. Dilatation of the pupil is more frequent on the contralateral side. Loss of pupillary reaction is again more frequent in hemorrhage.

If improvement is to follow cervical sympathetic block, it should be noticed within the next ten minutes. The improvement may then continue and should a relapse occur, the patient must be reinjected. We have not given these injections more often than once every twenty-four hours, but they might be given even eight to twelve hours apart if improvement is followed by signs of deterioration (Table 2).

A total of 105 injections have been given to 50 patients. More than half of these patients received only one injection, since in our early experience we were not sufficiently aware of the importance of reinjecting the patient if the first injection seemed useful. Today it is customary to inject the cervical sympathetics daily until no further improvement is noted. It also seems that bilateral sympathetic block is more effective, at least in the animal experiment, but no simultaneous injections have been made in this series.

TABLE 3

RESULTS CORRELATED WITH THE TIME ELAPSED BETWEEN ONSET AND STELLATE BLOCK

	NUMBER OF PATIENTS	IMPROVEMENT
First hour	5	4
2 to 6 hours	8	6
6 to 12 hours	7	5
12 to 24 hours	5	3
24 to 48 hours	20	3
More than 48 hours	5	0

THE TIME FACTOR

Gradually we have come to recognize the fact that, at least in the big cities, the patient must actually be in the hospital for some other reason when the cerebrovascular accident occurs so that the injection can be given immediately. Of the 5 patients injected within the first hour, 1 was a visitor, 1 a blue baby who developed a hemiplegia during the exposure of the tetralogy of Fallot, 1 was in the hospital for a coronary occlusion, and 2 had had previous peripheral emboli and were fibrillating (Table 3).

DISCUSSION

IN SPITE of the theoretical considerations which made it seem unlikely that the temporary elimination of vasomotor tonus of the cerebral vessels would effectively influence the deranged cerebral circulation following apoplexy, our clinical observations leave no doubt that in properly selected cases injected at the earliest possible moment, an acceleration of the phase of restitution may be expected. By this we mean that in all cerebrovascular accidents, surrounding the ischemic or hemorrhagic infarct, there exists a zone of stasis, vasoparalysis, and exudation of plasma. This has been seen in the experimental animal through a window in the skull by Villaret and Cachera. They also demonstrated the development of a collateral network around the infarcted areas and the existence of segmental spasms and dilatations in the nonobstructed vessels. They showed that these functional changes must be activated by an irritative sensory lesion of the obstructed vessels, since air-embolism, while producing complete vascular block, never was accompanied by such phenomena. Their observations, however, relate to the vessels of the pia which are known to have more vasomotor control than those of intracerebral vessels.

A second observation, which indicates the widespread cerebral damage a great distance from the original lesion, can be found in the histologic studies of Evans and Scheinker who found diffuse cerebral atrophy accompanying focal injuries in head trauma. They reported that the white matter stands the collateral edema and stasis much more poorly and that the hypoxia here is followed by diffuse gliosis. While recently there has been a tendency to minimize the importance of cerebral swelling in concussions, the accurate measurement of White, Brooks, Goldthwaite, and Adams showed that edema and congestion are present, due to extravasation of fluid through permeable anoxic capillary membranes.

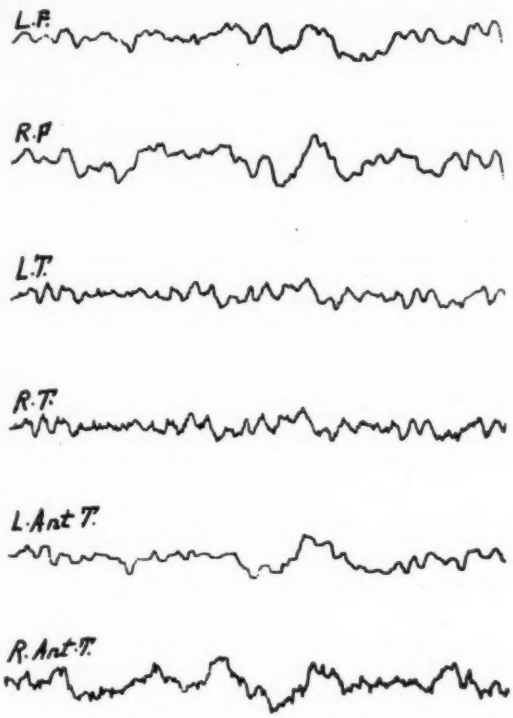

Figure 1. Electro-encephalogram report, St. Luke's Hospital, May 27, 1948. Male, 76 years of age; clinical diagnosis, left hemiplegia. Record shows irregular slow wave focus of 1½ to 2/seconds frequency in the right temporal, right anterior temporal, and bifrontal leads. Background activity about 7/seconds. Impression: Slow wave focus centering in the right frontotemporal area.

The question then arises, can sympathetic block relieve such stasis by (1) releasing the arteriolar spasm proximal to it; (2) improving venous drainage and thereby also decreasing intraventricular and intraspinal pressure, or (3) by so affecting the extracerebral portion of the carotids that more blood may be brought to the brain. Such studies are now under way.

Additional evidence that sympathetic block radically changes the function of the brain following a vascular accident is provided by electro-encephalograms taken before and fifteen minutes after a successful block in a case of cerebral thrombosis (Figures 1 and 2). It will be noted that the slow foci in the left frontal, right frontal, right temporal, and right anterior temporal leads have all been accelerated and have taken on a normal pattern.

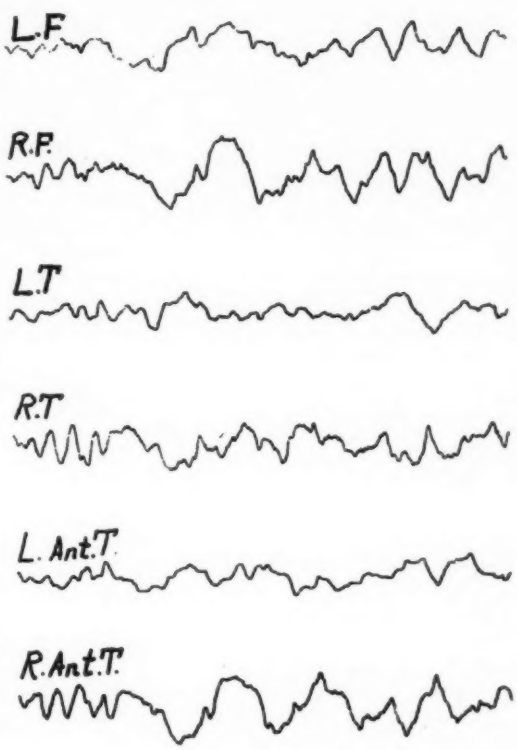

Figure 2. Electro-encephalogram report, St. Luke's Hospital, May 27, 1948. Male, 76 years of age. All slow activity decreased. Focus reduced and entire record looks more normal. Impression: greatly improved. (Tracing obtained immediately after stellate nerve block on right side of neck. Horner's syndrome present in right eye.)

TREATMENT

On the basis of our conception, the following therapeutic measures seem indicated in acute cerebrovascular accidents.

In patients with cerebral embolism, an oxygen tent, medication for slowing of rapid fibrillation, stellate block, and anticoagulants are ordered. Dicumarol may be kept up for weeks and months according to a schedule we have discussed elsewhere.

In patients with cerebral thrombosis, an oxygen tent, venesection in case of hypertension, stellate block, and release of increased spinal fluid pressure are indicated.

In patients with cerebral hemorrhage, an oxygen tent, slow spinal drainage, and neurosurgical consultation for possible evacuation of clots may be considered. No sympathetic block need be done.

In all three varieties of apoplexy, hypertonic sucrose or concentrated albumin with 5 to 7½ gr. (0.24-0.5 gm.) of aminophylline given intravenously should improve the cerebral edema. There is some evidence that aminophylline lowers spinal fluid pressure and its use seemed to us more rational than the extreme dehydration advocated in patients with cerebrovascular accidents or with head trauma.

ADDITIONAL MEASURES

Pulmonary ventilation—An embarrassed cerebral circulation is greatly handicapped if the oxygen exchange in the lungs is diminished. It is not enough, however, to place the unconscious or slightly cyanotic patient in an oxygen tent and leave him in the half-sitting position. Mucus and bronchial secretion collect in astonishing quantities in the tracheobronchial tree of the apoplectic patient, just as in head trauma. It is likely that, just as in pulmonary embolism and in reflex pulmonary atelectasis following chest trauma, this wet lung is the result of vagal stimulation which here is of central origin.

Since the majority of patients who survive the first forty-eight hours of the apoplectic insult die

of pneumonia, postural drainage of the tracheobronchial tree is an important and much-neglected measure in the unconscious patient. The patient is placed on his side, rather than on his abdomen, and the foot of the bed is slightly elevated. Mucus may be aspirated from the mouth and pharynx through a No. 14 French catheter. A metal airway may be inserted if necessary. Aminophylline and atropine may be used if secretion is excessive. It is astonishing how good postural drainage and some pharmacologic inhibition of bronchial obstruction and secretion may clear up neurologic signs due to anoxia and prevent the greatest single cause of mortality.

Nutrition—If after twenty-four hours the patient is still unconscious, parenterally administered fluids are substituted by hourly feeding through a Levine tube, keeping the patient's protein and vitamin requirements in mind. Dehydration, as practiced so extensively at one time in cases of head injuries, need not be carried out, and a fluid intake of 2,000 to 3,000 cc. of liquids should be maintained. Twenty-five per cent albumin intravenously gives excellent nourishment and serves a more useful measure against the wet brain than glucose or sucrose injections.

Urinary drainage—Overdistention of the bladder in the stuporous patient should not be allowed to persist; both retention and incontinence are encountered and these should be treated according to present-day urologic principles. The wet soiled linen greatly contributes to the occurrence of decubitus ulcers.

Sedation—Stuporous or unconscious patients require no sedation. Restless patients may have phenobarbital 0.6 to 0.2 gm. (1 to 3 gr.), bromides 1.2 to 2.4 gm. (20 to 40 gr.), chloral hydrate 1.2 to 2.0 gm. (20 to 30 gr.), or paraldehyde 4 to 12 cc. (1 to 3 drams) per rectum. For patients with severe restlessness or delirium, intravenous pentothal or amytal may be needed but narcotics are to be avoided for several reasons. Their miotic effect prevents the development of localizing eye signs, they depress the activity of the respiratory and cardiovascular mechanisms, they increase intracranial tension, they may exert undesirable side effects on the gastrointestinal tract, and finally as sedatives they do not calm the patient any more than the above mentioned less noxious agents. Generally speaking the apoplectic patient should have the benefit of treatment developed by neurosurgeons in cases of head trauma. These principles have not been consistently applied to the patient suffering from strokes.

Surgical exploration in apoplexy—This method of attack is still in its infancy. As Aring pointed out, every massive cerebral hemorrhage above a certain size is fatal. Our mortality in the present series is 77 per cent and here is a neurosurgical problem which needs to be attacked before the blood reaches the intraventricular system. Ventriculograms and arteriograms may be helpful in localizing the hemorrhage. The indications and results of such clot evacuations in cases of spontaneous intracerebral hemorrhage are yet unknown.

SUMMARY

Attention has been called to the importance of a differentiation between cerebral embolism, thrombosis (or softening), and hemorrhage. The importance of collateral stasis, vasoparalysis, and edema, accompanying the cerebrovascular accident has been stressed. In 50 patients the cervical sympathetic trunk was blocked with procaine on the side of the apoplexy. Good response was obtained in 41 of these. Regaining of consciousness, of speech, motor improvement, and conversion of flaccid into spastic paralysis were also observed. Other methods of combating cerebral edema and stasis may be added. A less passive attitude in the treatment of apoplexy is suggested.

SALMON MEMORIAL LECTURER NAMED

DR. Stanley Cobb has been named the Salmon Memorial Lecturer for 1949 by the Salmon Committee on Psychiatry and Mental Hygiene, appointed by the New York Academy of Medicine. Dr. Cobb is Bullard professor of neuropathology at Harvard Medical School, a post he has held since 1926. He has been psychiatrist in chief of the Massachusetts General Hospital for the past fifteen years. Every year since 1932 the Salmon Committee has chosen as lecturer an outstanding specialist in the field.

Public Understanding of the Vitamins

ROBERT S. GOODHART*

NEW YORK

THERE is a great deal that we do not know about the vitamins, their methods of action within the body, and the metabolic dysfunctions associated with their absence or deficiency. There is ample information, however, to permit physicians and the general public to evaluate intelligently the probable importance of the vitamins in their daily diets and in therapeutics.

Much of the confusion that exists in the minds of many people appears to arise from the failure to comprehend the basic nature of the vitamins as essential nutrients which do not themselves produce energy or body tissues but without which neither energy nor tissue can be manufactured by the living body. Those vitamins for which specific biochemical functions have been found, e.g., thiamine, riboflavin and niacin, have been shown to act within the body as components of enzyme systems essential for the metabolism of fats, carbohydrates, and proteins, as components of mechanisms necessary for the transport of fat and as sources of labile methyl groups (choline), or as essentials for the formation of other specific biologic substances (vitamin K and prothrombin, vitamin A, and rhodopsin). General understanding of the sparking and mediating roles of the vitamins in metabolism would do much toward resolving popular misconceptions and the frequently senseless arguments about the relative merits of natural foods and the synthetic vitamins.

The vitamins are useless without substrata upon which to act or media in which to operate, without carbohydrates, protein, fat, water, oxygen, and certain minerals. The proper foods are essential for health and the vitamins, either natural or synthetic, are essential for the efficient and optimal utilization of ingested foodstuffs by the body.

It is true that natural foods contain, in addition to other essentials, a number of vitamins and vitamin-like substances which are not available in the synthetic form. It is also true that most of the foods eaten by the average American are not natural foods in the sense that they contain all of the nutrients of the freshly harvested, mature products. Most of the foods which we eat have suffered losses of important dietary essentials through premature harvesting and through storage, processing, kitchen preparation, cooking, and serving. Thus, although it is important that we choose our foods wisely, according to the approximate content of essential nutrients, it is not always

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wise to rely upon so-called natural foods for all of our vitamin requirements.

The fact that there may be some vitamins essential for man which are not available in synthetic form or in vitamin concentrates does not constitute a valid argument against the use of the synthetic vitamins as dietary supplements for man. It is probable that the vitamins now available in synthetic or concentrated form include all of those of which mankind is ever likely to suffer a deficiency of primary dietary origin, with existing dietary customs. No primary dietary deficiency disease in man has been ascribed to any still unidentified vitamins in food. If any of these unidentified substances are dietary essentials for man, as they may well be, either they are needed in very small amounts or they are widely distributed in foods and are resistant to destruction by common methods of food preparation. There is absolutely no evidence that one, two, three, or four times the maintenance amounts of the synthetic vitamins has ever produced any deleterious effects when used as an oral supplement to the diet of man, even over extremely long periods of time.

It is the popular practice for nutritionists to advise lay individuals to consult their physicians before resorting to vitamin supplementation of their diets. Why this should be when, at the same time, the same nutrition authorities are entirely uninhibited in their promotion of vitamins and other nutrients as they occur in edible farm products is a problem to be resolved by the moralist. The plain fact is that there is not a single practicing physician who can determine the precise nutritional requirements of any one of his patients or who can say with certainty that this or that one of his patients is enjoying optimal health.

There are many reasons why routine medical check-ups for everyone may be desirable and certainly individuals who consider themselves to be ailing should be encouraged to consult their physician. But, if supplementation of the diet with non-therapeutic amounts of the synthetic vitamins is to be the responsibility of the physician and of no one else, then the responsibility for the correction of all dietary problems and for nutrition education belongs to him and to no one else (and I am not saying that this would be a bad state of affairs).

THE requirements for particular vitamins are affected by composition of a person's diet, his energy intake and expenditure, his state of physical maturity, the presence of diseases, trauma, or metabolic conditions which increase requirements or interfere with the absorption or utilization of nutrients, probably by genetic factors which may account for some observed differences between the requirements of individuals and possibly by physiologic adaptations to customary intakes. Obviously a single figure cannot be taken as representing the exact requirement of all persons of a given age and state of health for any particular vitamin (or other nutrient). Because of this fact and in order to help assure

the great majority of the people in this country the possibility of obtaining an adequate diet, The Food and Nutrition Board of The National Research Council[1] examined all of the available experimental data and prepared a table of Recommended Dietary Allowances (Table 1) designed to meet "the immediate needs of normal persons with relatively low efficiency." It is recommended that physicians use this guide in estimating the adequacy of the diets of "healthy" persons, bearing in mind that some individuals will require more than the recommended amounts of certain nutrients and that the Recommended Dietary Allowances refer to nutrients actually ingested and not as present in foods when purchased.

ALL DIETARY surveys in this country indicate a marked prevalence of dietary inadequacies as judged by the Recommended Dietary Allowances.[2] For the reasons indicated above, this is not synonymous with the prevalence of nutritional deficiencies.

The anatomic and biochemical criteria presently advocated and used in nutrition surveys for the determination of the prevalence of mild and chronic vitamin deficiency states are unsatisfactory. Very few of the anatomic lesions have been proved to be pathognomonic of specific vitamin deficiencies and in the production of some of them other traumatic factors may be of major importance. Serious functional disturbance may precede the development of objective anatomic signs by a considerable period of time and may be present when casual determinations of blood and urine vitamin content show levels within the normal range. Nevertheless these tests are useful when applied to large groups of the population, as indications of the probable prevalence of certain vitamin deficiencies. The results of chemical studies and of the medical assessment of nutritional status are in agreement with those of dietary surveys in suggesting a high incidence of mild and chronic vitamin deficiency in the population of the United States.[2]

The order of frequency of the common vitamin deficiencies appears to be about as follows: vitamin A, riboflavin, niacin, ascorbic acid, vitamin D, and thiamine. The order of frequency varies, of course, in different sections of the country, in different economic and social groups, and in different diseases. It must be remembered, also, that a deficiency of a single vitamin practically never occurs in man. Multiple deficiency is the rule and multivitamin therapy is the treatment of choice.

It cannot be overemphasized that prevention of the development of nutritional deficiencies is much more efficient, in terms of conserving the health and of prolonging the active, productive life of the individual, than periodic attempts to correct nutritional deficiency states. Therapeutic amounts of the vitamins are five to ten times greater than optimum maintenance amounts and therapy generally must be continued over months and even years. Some of the anatomic lesions of vitamin deficiency are not subject to correction. If the age of onset of senescence is influenced at all by nutritional status, the initial effect occurs early in life and proper dietary habits should be inculcated early in childhood.

The water-soluble vitamins are not well stored by the body and in order to insure good nutrition the daily intake of them should be adequate. In all disease states where the requirement is increased, or the absorption or utilization is decreased, the vitamin intake should be increased. Periodic deficiencies may be the cause of chronic abnormalities.

It is theoretically possible to prevent the development of any degree of vitamin deficiency in a well individual by dietary means alone. It seems to me that this would be extremely unlikely in the presence of disease and I doubt that any individual goes through life without at some time or other developing a vitamin deficiency, the scars of which he carries to his grave.

It is senseless to attempt therapy of vitamin deficiency states with "natural" foodstuffs alone, when the synthetic vitamins and vitamin concentrates are available. It is not fair to the patient to deny him therapeutic amounts of these

TABLE 1

RECOMMENDED DIETARY ALLOWANCES, REVISED 1945[1, 2]

(Amounts Per Day)

FOOD AND NUTRITION BOARD, NATIONAL RESEARCH COUNCIL

	CALORIES	PROTEIN GRAMS	CALCIUM GRAMS	IRON MG.
Man (154 lb., 70 kg.)				
Sedentary	2,500	70	0.8	12[4]
Moderately active	3,000	70	0.8	12[4]
Very active	4,500	70	0.8	12[4]
Woman (123 lb., 56 kg.)				
Sedentary	2,100	60	0.8	12
Moderately active	2,500	60	0.8	12
Very active	3,000	60	0.8	12
Pregnancy (latter half)	2,500[6]	85	1.5	15
Lactation	3,000	100	2.0	15
Children up to 12 yrs.[7]				
Under 1 yr.[8]	100/2.2 lb. (1 kg.)	3.5/2.2 lb. (1 kg.)	1.0	6
1-3 yrs. (29 lb., 13 kg.)	1,200	40	1.0	7
4-6 yrs. (42 lb., 19 kg.)	1,600	50	1.0	8
7-9 yrs. (55 lb., 25 kg.)	2,000	60	1.0	10
10-12 yrs. (75 lb., 34 kg.)	2,500	70	1.2	12
Children over 12 yrs.[7]				
Girls, 13-15 yrs. (108 lb., 49 kg.)	2,600	80	1.3	15
16-20 yrs. (119 lb., 54 kg.)	2,400	75	1.0	15
Boys, 13-15 yrs. (103 lb., 47 kg.)	3,200	85	1.4	15
16-20 yrs. (141 lb., 64 kg.)	3,800	100	1.4	15

[1]Tentative goal toward which to aim in planning practical dietaries; can be met by a good diet with a variety of natural foods. Such a diet will also provide other minerals and vitamins, the requirements for which are less well known.

[2]The allowance depends on the relative amounts of vitamin A and carotene. The allowances of the table are based on the premise that approximate two-thirds of the vitamin A value of the average diet in this country is contributed by carotene and that carotene has half or less than half the value of vitamin A.

[3]For adults (except pregnant and lactating women) receiving diets supplying 2,000 calories or less, such as reducing diets, the allowances of thiamine, riboflavin, and niacin may be 1 mg., 1.5 mg., and 10 mg. respectively. The fact that figures are given for different calorie levels for thiamine, riboflavin, and niacin does not imply that we can estimate the requirement of these factors within 500 calories, but they are added merely for simplicity of calculation. Other members of the B complex also are required, though no values can be given. Foods supplying adequate thiamine, riboflavin, and niacin will tend to supply sufficient of the remaining B vitamins.

[4]There is evidence that the male adult needs little or no iron. The allowance will be provided if the diet is satisfactory in other respects.

TABLE 1

VITAMIN A I.U.[2]	THIAMINE MG.[3]	RIBOFLAVIN MG.[3]	NIACIN (NICOTINIC ACID) MG.[3]	ASCORBIC ACID MG.	VITAMIN D I.U.
					(No. refers to footnote)
5,000	1.2	1.6	12	75	5
5,000	1.5	2.0	15	75	5
5,000	2.0	2.6	20	75	5
					(No. refers to footnote)
5,000	1.1	1.5	11	70	5
5,000	1.2	1.6	12	70	5
5,000	1.5	2.0	15	70	5
6,000	1.8	2.5	18	100	400 to 800
8,000	2.0	3.0	20	150	400 to 800
1,500	0.4	0.6	4	30	400 to 800
2,000	0.6	0.9	6	35	400
2,500	0.8	1.2	8	50	400
3,500	1.0	1.5	10	60	400
4,500	1.2	1.8	12	75	400
5,000	1.3	2.0	13	80	400
5,000	1.2	1.8	12	80	400
5,000	1.5	2.0	15	90	400
6,000	1.8	2.5	18	100	400

[5]For persons who have no opportunity for exposure to clear sunshine and for elderly persons, the ingestion of small amounts of vitamin D may be desirable. Other adults probably have little need for vitamin D.

[6]During the latter part of pregnancy the allowance should increase approximately 20 per cent over the preceding level. The value of 2,500 calories represents the allowance for pregnant, sedentary women.

[7]Allowances for children are based on the needs for the middle year in each group (as 2, 5, 8, etc.) and are for moderate activity and for average weight at the middle year of the age group.

[8]Needs of infants increase from month to month with size and activity. The amounts given are for approximately 6 to 8 months. The dietary requirements for some of the nutrients such as protein and calcium are less if derived largely from human milk.

[9]The requirement for vitamin K usually is satisfied by any good diet. Special consideration needs to be given to newborn infants. Physicians commonly give vitamin K either to the mother before delivery or to the infant immediately after birth.

substances when he presents signs and symptoms known to be associated with vitamin deficiency states. The physician has a responsibility to keep himself acquainted with the positive and suggestive signs and symptoms of mild and chronic vitamin deficiency states. Nutrition is not merely a branch of medicine, it is a foundation stone for health.

SUMMARY

The vitamins are neither dangerous drugs nor panaceas. They are accessory food substances necessary for the metabolism of other foodstuffs and the discharge of important body functions. They are of no value to the body in the absence of a substratum upon which to act and the synthetic vitamins cannot be substituted for customary foods.

Most farm products eaten by civilized man have undergone changes since harvesting and should not be considered to be "natural." Among other nutrients, certain vitamin losses occur in harvested foods by the time they are eaten. To compensate for these losses by the vitamin fortification of important staple foods or by the use of polyvitamin preparations constitutes a sensible and desirable procedure in preventive medicine.

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THE FIRST MEDICAL EDITOR

(From: "The Medical Pickwick," a monthly literary magazine which suspended publication about 1920.)

DIAGNOSTIC CLINIC

Antipernicious Anemia Substances

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Liver extract or folic acid or both are effective in only a definitely limited number of conditions, namely, certain of the macrocytic anemias which are associated with megaloblastic hyperplasia in the marrow, by which we mean that the marrow contains very immature cells of the red cell series. This group of diseases includes pernicious anemia, which is by far the most common type of this anemia seen in this part of the country. It also includes sprue, both the tropical and nontropical varieties, of which, of course, we see very little here; certain macrocytic anemias of pregnancy which are not common in my own experience; certain nutritional deficiency anemias which produce anemia similar in appearance to that of pernicious anemia and which Dr. Tom Spies describes, particularly in people living in the South; and, finally the macrocytic anemia of infancy, usually seen in children under 2 years of age, and particularly in those children who have been on diets containing milk substitutes.

It is important to note at least theoretically that all these anemias which do respond either to liver extract or folic acid are associated with megaloblastic hyperplasia in the marrow. It does not mean that one necessarily has to demonstrate this in each of his patients, but it does mean that one must know that type of disorder is present in the marrow, because in the absence of the megaloblastic hyperplasia in the marrow these therapeutic materials are not effective.

DIAGNOSIS OF PERNICIOUS ANEMIA

So far as the diagnosis of pernicious anemia is concerned (and I should like to devote most of my discussion to pernicious anemia because it is the most common type of these anemias), it is, of course, necessary to do certain laboratory procedures in addition to a history and physical examination. These, however, are not so complicated that they cannot be done by any physician who has facilities for performing a red blood count and hemoglobin determination. We find a reduction in the red blood cell count and hemoglobin, in addition to which there is almost uniformly, in pernicious anemia in relapse, a reduction in the white blood count and the number of blood platelets.

One thing that is useful in making a diagnosis of pernicious anemia is demonstrated in Figure 1, in which we see the various blood indices. The mean corpuscular volume expresses the volume of the erythrocyte, the mean corpuscular hemoglobin gives the weight of hemoglobin in the red cell, and the mean corpuscular hemoglobin concentration shows the percentage of the cell which is hemoglobin. It is not necessary to figure out all of these for the clinical diagnosis of pernicious anemia.

I should like to call your attention particularly to the color index. You are all familiar with this, I am sure. It is calculated by dividing the *percentage* of hemoglobin by the *percentage* of the red blood count. In order to express a value in *per cent,* something has to be considered a hundred *per cent,* which makes this a little bad, but for ordinary purposes in figuring the color index we assume a red blood count of 5,000,000 per cu. mm. to be 100 *per cent* of normal and a hemoglobin of 15 or 15.6 gm. per 100 ml. to be 100 *per cent* of normal. If one then had a patient with a red blood count of 5,000,000 and a hemoglobin of 15.6 gm., the color index would be $\frac{100}{100}$ or 1. So normally the color index is unity.

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In pernicious anemia the color index is elevated, which means that each of the red blood cells is carrying a greater than normal amount of hemoglobin. For example, one might find a pernicious anemia patient with a red blood count of, let us say 2,000,000, which is 40 per cent of normal, but instead of the hemoglobin being 40 per cent it might be 60 per cent of normal, so that the color index would become $\frac{60}{40}$, a color index of 1.5, or greater than unity.

We can tell by looking at the red blood cells on the smear that something is changed. Some normal red blood cells (Figure 2) may be compared with red blood cells of pernicious anemia (Figure 3). The normal cells are more or less uniform in size and shape and in their staining reaction. The white cell is a lymphocyte.

When pernicious anemia is present, the cells tend to be larger. There are many large cells, as can be seen in Figure 3. You can see, however, that there are also small cells, so that there is considerable variation in size of the red blood cells. Then you observe the odd-shaped cells such as a sickle cell, a tear-drop shape, and others. In pernicious anemia there is an increase in size of the red blood cells and a variation in the size and shape of the cells.

In addition to these things, it is necessary, in order really to establish a diagnosis of pernicious anemia, to do a little more laboratory work. One of the things that should certainly be done is a gastric analysis. Patients with pernicious anemia are lacking in gastric hydrochloric acid. If one finds hydrochloric acid in the stomach of a patient, one can be sure that pernicious anemia is not present. The converse is not necessarily true. That is to say, some normal people do not have free hydrochloric acid in the stomach, but no patient with pernicious anemia has gastric hydrochloric acid. In order to be sure of this it is necessary to give the patient histamine.

Histamine is one of the most potent stimulators of gastric secretion of which we know. We give the patient 0.5 mg. or 1.0 mg. of histamine subcutaneously and see if there is any acid present in the stomach twenty or forty minutes after such administration. In pernicious anemia there is no free hydrochloric acid after histamine is administered. It is very important that this test be performed if you decide you are going to make a diagnosis of pernicious anemia, because if there is hydrochloric acid in the stomach, that patient does not have pernicious anemia and it means that you do not have to treat the patient the rest of his or her life with liver extract. Once you make a diagnosis of pernicious anemia you are committed to treat this patient regularly the rest of his life. It is a shame to do that if true pernicious anemia is not really present.

For clinical purposes I think that is enough laboratory work. In other words, you demonstrate that an anemia is present; you demonstrate that the red cells are large by noting what the color index is and by looking at the red blood cells on the smear; and you demonstrate that the patient has no acid. Having done that, you are certainly justified in making a working diagnosis of pernicious anemia and seeing what happens with treatment.

Figure 4 shows what does happen when a patient with pernicious anemia is treated with an active material, which in this case was folic acid. When a patient is treated with any active material, either liver extract or folic acid, one of the first changes is a rise in reticulocytes. The chart shows daily reticulocyte counts recorded. It is not absolutely necessary to do daily reticulocyte counts, because while it is true that the reticulocytes go up

$\frac{\text{Vol. (cc. per 1,000 cc.)}}{\text{R.B.C. (in millions)}}$	= corpuscular volume in c.μ	$\frac{\text{Vol. per cent}}{\text{R.B.C., per cent}}$	= volume index
$\frac{\text{Hb. (gm. per 1,000 cc.)}}{\text{R.B.C. (in millions)}}$	= corpuscular hemoglobin in 10-12 gm.	$\frac{\text{Hb., per cent}}{\text{R.B.C., per cent}}$	= color index
$\frac{\text{Hb. (gm. per 100 cc.)}}{\text{Vol. (cc. per 100 cc.)}}$	= proportion of hb. in cell;	$\frac{\text{Hb., per cent}}{\text{Vol., per cent}}$	= saturation index

Figure 1. Blood indices useful in making diagnosis of pernicious anemia as explained on page 197.

within five to ten days to a very high peak, if you want to wait just a few more days you will find that the red blood count eventually goes up. In this case there was a definite increase in the red blood count at the end of twenty-five days. The hemoglobin likewise goes up. The white cells, which were a little low here, go up and finally stay up. So while it is nice to have daily reticulocyte counts, it is not absolutely necessary.

One thing I do want to point out to you very definitely (and I would like to emphasize this strongly) is that if a patient has pernicious anemia, the red blood count will be approximately normal within sixty days after the start of adequate treatment. That is important, because if the red blood count has shown very little change by the end of some sixty days the diagnosis of pernicious anemia is doubtful. A true case of pernicious anemia will have had a rise in the red blood count and the hemoglobin to normal within sixty days.

In this case, after some fifty-three days, the red blood count was at a level of 4,000,000, the hemoglobin at a level of over 80 *per cent*. If the patient does not respond within that approximate length of time you have reason to feel that the diagnosis of pernicious anemia is in error.

If you want to do more in the way of diagnosis than this, you may. There are a couple of other things that are done, usually by people who are investigating cases and reporting experiments in the literature. One of these is a bone marrow examination. It is nice to have it. One can do a sternal puncture and demonstrate in the aspirated material the presence of the megaloblasts, the abnormal and immature cells which are present in the marrow of patients with pernicious anemia.

The other thing that we do in working up these cases thoroughly for case reports is the determination of the complete indices shown in Figure 1. Again I emphasize that it is not necessary to have all of these indices in making a clinical diagnosis of pernicious anemia. Also if you can do daily reticulocyte counts and find a marked increase in reticulocytes within five to ten days, that is a quick way of telling yourself that the patient is responding to treatment.

TREATMENT OF PERNICIOUS ANEMIA

So far as the treatment of pernicious anemia is concerned, liver extract is still the treatment of choice. Considering first the patient with pernicious anemia in relapse, we find that we can treat these people with either oral or intramuscular therapy. For oral therapy we have liver extract and stomach concentrate and mixtures of the two. Very few patients are now being treated with these materials, for two reasons. One is that absorption is uncertain in these people and, therefore, you will find a certain number of patients with pernicious anemia who do very poorly on oral medication. Their red blood counts do not come up well; they do not stay up well because of the poor absorption which takes place in the gastrointestinal tract. The other reason is that the patient who is receiving oral medication cannot always be depended upon to take it. That is particularly true of the patients with pernicious anemia. When they get to feeling well, as indeed they do after they have been adequately treated, some of them are reluctant to believe that they are any longer sick and do not want to take any more medicine. Consequently we find that the oral therapy of pernicious anemia is not commonly used, and parenteral therapy is the common method of treatment.

For a patient in relapse, we ordinarily give a great deal more liver than is necessary. If the patient is in the hospital it is common practice to give an injection of liver extract daily. You do not have to, but, on the other hand, you cannot give too much liver extract. If the patient is not sick enough that he needs to be in the hospital, an injection once or twice a week is adequate.

We use, in these days, the so-called purified liver extracts. These are liver extracts which contain from 5 to 15 antipernicious anemia units per milli-

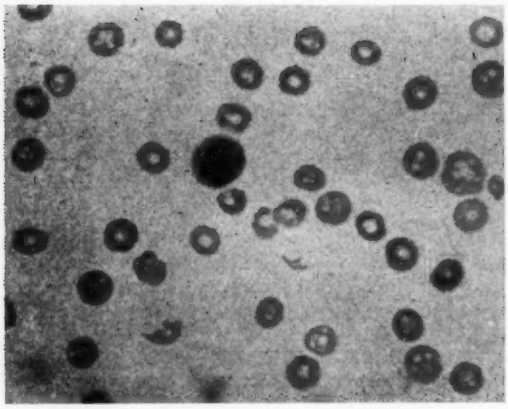

Figure 2. Normal red blood cells.

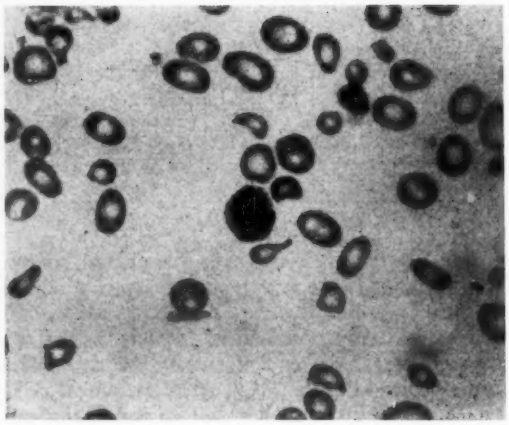

Figure 3. Red blood cells of pernicious anemia.

liter, and I think that there is no reason not to use the 15-unit material. As a matter of fact, most of the 10- and 5-unit materials are 15-unit materials which have been diluted with saline, so that you are paying for more bottles and some salt solution, and so on, and you might just as well use the 15-unit material to start with. If the patient is in the hospital you can administer 1 ml. containing 15 units daily intramuscularly. If the patient is not in the hospital, give 15 units once or twice a week until such time as the red blood count has reached approximately normal. I like to administer the liver extract at least once a week until the red blood count has reached approximately normal, which means a period of about sixty days.

The problem of the treatment of the patient in remission also arises. Now that the anemia has been relieved, and the red blood count is more nearly normal, it is desirable to start to spread the intervals between injections of liver extract. In my own practice I ordinarily give it every couple of weeks for a couple of months and then every three weeks for two more months, and finally put the patient on a schedule of receiving liver extract every four weeks. Most patients will do well on such a regimen. When you arrive at this interval of treatment, 2 ml. of the 15-unit material or 30 units every four weeks is sufficient liver extract therapy for practically all patients. I do not believe that one should make the interval greater than four weeks, because liver extract is not stored quantitatively, and if one starts stretching the interval to six weeks, eight weeks, and so on, many of these patients will not maintain good red blood counts, no matter how large the individual dose of liver extract. In other words, interval is just as important as dose, perhaps more so. A good working rule is not to make the interval between injections of liver extract greater than four weeks.

USE OF FOLIC ACID

About three years ago, folic acid was first described in the treatment of the macrocytic anemias. Most patients with pernicious anemia will respond to folic acid. Their blood counts will go to normal and can be maintained at normal in most cases. When this was discovered, it appeared that folic acid was a very desirable drug, because it is highly effective when taken by mouth in relatively small amounts, and we thought that perhaps we had a considerably better method of treatment of pernicious anemia than with liver extract. However, we now know that folic acid cannot be considered as the proper method of treatment of pernicious anemia, for a number of reasons.

THE course of a patient with pernicious anemia who did not respond to folic acid when we started treatment during relapse is shown in Figure 5. Here was a patient who had a red blood count of just a little over 1,000,000. We started folic acid in doses of 1 mg. a day given intramuscularly. There

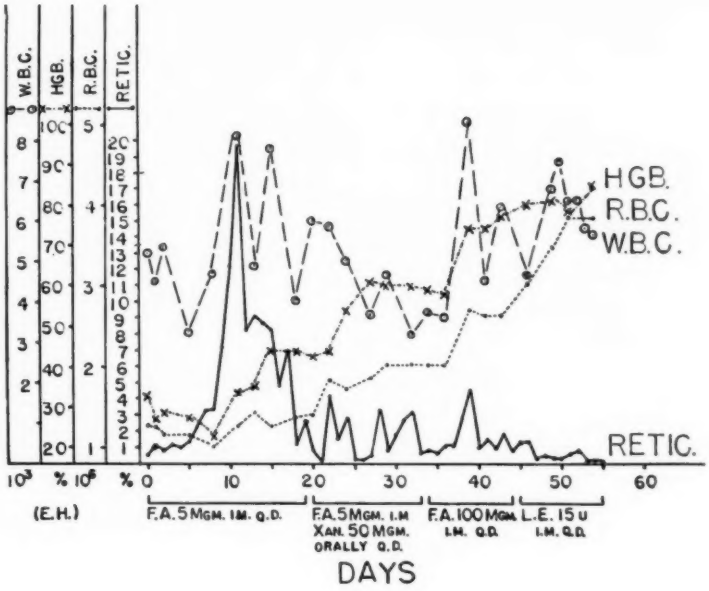

Figure 4. Daily reticulocyte counts in patient with pernicious anemia treated with folic acid.

was a very late delayed reticulocyte response, which was not nearly as big as it looks on the chart; actually it was only about *4 per cent,* whereas one would have expected a reticulocyte response nearer *30 per cent.* During the second period we raised the dose to 6 mg. a day. We got another very delayed and small reticulocyte response. The dose was raised to 12 mg. during the third period, with possibly another small delayed response. During a fourth period we continued the folic acid with some xanthopterin. Sufficient to say that within some forty-three days of folic acid therapy the patient showed very little improvement. The red blood count and hemoglobin rose only very slightly during that time.

When we gave this patient 15 units of liver extract daily there was a prompt and good reticulocyte response and a very rapid increase in the red blood count and hemoglobin.

Most patients with pernicious anemia do not react in this way but will respond to folic acid, but some of them will not and this is a demonstration of one that would not.

What about the maintenance of pernicious anemia with folic acid? Suppose that we get the red blood count and hemoglobin up to normal, can we maintain them on folic acid therapy? The answer is, not in all cases.

Table 1 shows what happened to a group of patients who were treated in our outpatient department with folic acid. You will notice that of 32 patients treated, 16 had no change in the red blood count or hemoglobin when they were treated with folic acid instead of liver extract, but two had a moderate decrease of from 300,000 to 500,000 per cu. mm. in the red count and seven had a rather marked decrease of over half a million. In other words, 9 of the 32 patients did not do well on folic acid. Why is that so? When we examine the data (Table 2), we find that in the nine relapses which occurred, five were in patients who were receiving individual doses of 40 to 50 mg. at intervals of three to four weeks, and four were getting a bigger dose, 150 mg., at intervals of three to six weeks. This demonstrates that one cannot give folic acid at intervals longer than two weeks. If given at intervals of two weeks or less or in daily doses no relapses occurred in this series but when we spread the interval to longer than two weeks, we found that some of the patients could not maintain normal blood levels.

In addition, we have found during the last couple

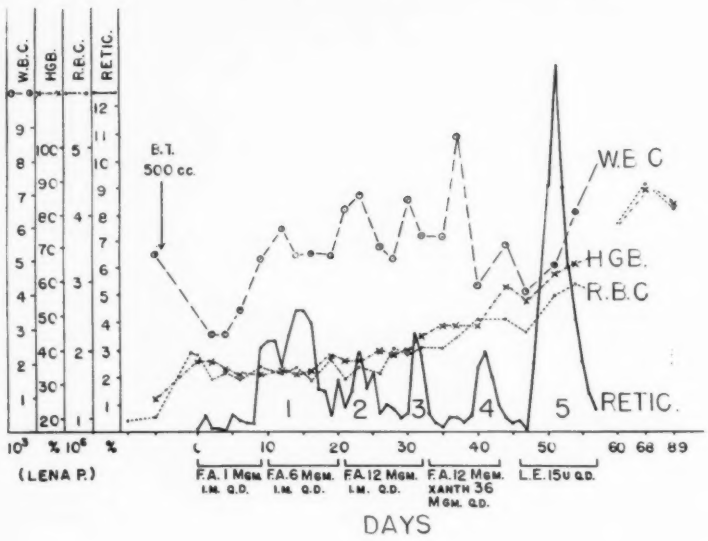

Figure 5. Course of patient with pernicious anemia who did not respond to folic acid treatment started during relapse.

of years that even though we give folic acid daily, we cannot maintain the blood level in all cases. Finally the patients no longer respond to folic acid, they begin to relapse, and their blood count drops. We can say with great certainty, therefore, that there are some patients with pernicious anemia who do not respond to folic acid when they are in relapse; there are other patients with pernicious anemia who cannot be maintained in hematologic remission with folic acid.

Another reason that we cannot use folic acid in treating pernicious anemia is illustrated in Figure 6. Here was a patient with pernicious anemia who was given 10 mg. daily of folic acid. There was a very nice reticulocyte response and a very rapid increase in the red blood count and hemoglobin, so that again at about the end of our sixty-day period the red blood count and hemoglobin were normal. At this point we raised our dose of folic acid because the patient very suddenly began to develop the neurologic disorder which is so much a part of pernicious anemia—he began to have numbness and tingling of the hands and feet. This progressed with extreme rapidity until in a few days he was completely numb from the waist down; his hands were completely numb, and he began to have difficulty in walking, and in general, had a severe neurologic relapse.

TABLE 1

PATIENTS TREATED 6 TO 12 MONTHS (AVERAGE 10.7)

No change in red count or hemoglobin	18
Moderate decrease in red count (300,000-500,000 per cubic millimeter)	2
Marked decrease in red count (over 500,000 per cubic millimeter)	7
Improvement in red count (310,000-1,120,000 per cubic millimeter)	3
Treated from relapse with satisfactory response	2
TOTAL	32

We raised our dose of folic acid for only ten days, which is not a long enough test period, but by that time he was so bad we were unwilling to wait any longer and instituted liver extract therapy. At the end of the time shown on this chart he was improving, but it took about six months of intensive liver extract therapy before recovery was complete. We now know that folic acid is completely unable to prevent the neurologic disorders which we see in pernicious anemia—the combined system disease of posterolateral column degeneration—and is completely unable to control it.

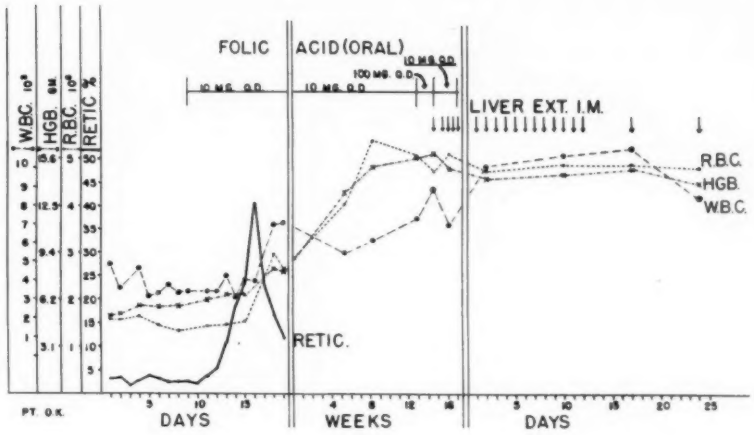

Figure 6. Course of pernicious anemia patient with good reticulocyte response to treatment with 10 mg. folic acid daily. Red blood count and hemoglobin were normal after sixty days, but patient then developed a neurologic disorder. Folic acid dosage was raised, but patient did not respond and liver extract therapy was instituted after ten days. Recovery was complete only after about six months of very intensive liver extract therapy.

WHAT about the combined use of folic acid and liver extract? I think that liver extract alone is satisfactory treatment and probably does not need folic acid added to it. There is some evidence, however, that if one does use folic acid in addition to liver extract, there may be a faster improvement in the blood and perhaps somewhat higher levels can be maintained.

There are on the market at this time liver extracts for parenteral injection which contain folic acid. I do not recommend these preparations because they contain relatively so little folic acid that it is not enough to make any difference. If you want to use combined therapy I recommend that you give parenteral liver extract at intervals as we have described and, in addition, give the patient one tablet of folic acid a day by mouth, 5 mg. a day.

I brought with me this morning one of my patients who is also a good friend of mine. This lady was first admitted to a local hospital in February 1945. At that time she was 54 years of age, and her main complaint was that of difficulty in walking. She told us that for two years she had had a gradual onset of staggering, numbness of the feet, and noted incoordination in performing moderately skilled acts.

When we examined her in the hospital we found that her gait was extremely ataxic; she had some spasticity of her lower extremities; there was loss of position sense in the lower extremities—you could move her toes up and down and she didn't know in which direction you were moving them. There was loss of vibratory sensation—she could not appreciate the vibrating tuning fork below the hips; all of her deep reflexes were hyperactive, that is, they were increased over normal; there was a Babinski reflex present bilaterally, and the Gordon and Hoffmann signs were also present. She demonstrated, therefore, a considerable degree of posterolateral column degeneration, the combined system disease seen in pernicious anemia. She was not very anemic. Her red blood count was 3,790,000; her hemoglobin was 12 gm. or 77 per cent by our scale; her white blood count was 4,200, below normal; her color index was a little bit over 1; her mean corpuscular volume was 106 cubic microns, which means that the cells were bigger than normal. She had no acid in her stomach after stimulation with histamine.

We made a diagnosis of pernicious anemia and recognized that the neurologic disorder was more severe than usual for the degree of anemia present. We treated her in this instance with a crude liver extract, although purified liver extract would have been entirely satisfactory. She received daily injections of 5 ml. of 2-unit liver extract all the time she was in the hospital. After she left the hospital she was treated with 30 units of purified liver extract weekly for four weeks, then 30 units every two weeks for three times, and then the same dose every four weeks up to the present time.

In sixty days her red blood count had risen to 4.76 million, the hemoglobin had risen to 15.6 gm. or 100 per cent, and the white count had come up

TABLE 2
FOLIC ACID MAINTENANCE

ROUTE	INDIVIDUAL DOSE	INTERVAL	NUMBER TREATED	NO CHANGE	RELAPSE	IMPROVEMENT
Intramuscular	45-50 mg.	2 weeks	2	0	0	2
Intramuscular	40-50 mg.	3-4 weeks	13	7	5	1
Intravenous	150 mg.	3 weeks	1	1	0	0
Intramuscular	150 mg.	3-6 weeks	9	5	4	0
Oral	10-40 mg.	Daily	16	13	0	3
TOTALS			41	26	9	6

to 6,350. There was marked improvement in gait and coordination. By October 1945, ten months after treatment was started, the patient told me one day she was even able to run a little bit. Since that time she has been able to walk perfectly normally; the abnormal reflexes have disappeared, and so far as the patient is concerned she is asymptomatic and in excellent health.

Although she did not look very sick at the time we saw her, it is of interest that her weight, which was 111 pounds at that time, has come up to its present 133 pounds.

This patient demonstrates one of the more unusual types of pernicious anemia patients where the neurologic symptoms are more outstanding than the anemia. Especially vigorous liver extract therapy is required when the neural disturbance is marked.

I ALSO want to mention briefly the other part of this subject, namely, the abuse of liver extract. If you are not absolutely certain that a patient has pernicious anemia but you think he may have, it is perfectly justifiable to give a therapeutic test with liver extract and it should be done as outlined for the patient with pernicious anemia as presented earlier. A trial of sixty days is long enough, and don't do what is so often done—give liver extract two to three times a week for a year or two or three years or longer.

If you are going to treat a patient who you suspect has pernicious anemia, use parenteral liver extract. Do not use mixtures of liver and iron which come in capsules. That is shotgun medication. You say, "Well, what if it is? If it is satisfactory, why shouldn't we use it?" There are two reasons. One is that if it does work you do not know which was effective, the liver or the iron, and you have no way of knowing which to use in continued treatment. Iron deficiency anemia is undoubtedly the most common type of anemia and you usually want to treat with iron. If you give the combined preparation, you give a preparation that is far more expensive than the simple iron medication.

If you have treated the patient who you suspect may have pernicious anemia with liver extract and, if within sixty days the red blood count has not shown the proper improvement, you must think of other diseases which can give a peripheral blood picture similar to that of pernicious anemia. These include, particularly, multiple myeloma or plasma cell myeloma; certain cases of renal insufficiency that have marked nitrogen retention and are going toward uremia will develop a macrocytic anemia; certain of the lymphomas, lymphosarcoma and Hodgkin's disease particularly, may at times be associated with an anemia which resembles pernicious anemia.

Finally, certain cases of carcinoma, particularly carcinoma of the stomach, may be associated with a macrocytic anemia. These anemias are not associated with megaloblastic hyperplasia in the marrow, so if you can get a marrow examination it will help you. If you cannot get a marrow examination all you have to remember is that if you have treated that patient for sixty days with liver extract and he has not responded, it is not pernicious anemia and you must therefore think of a different disease.

DIAGNOSTIC CLINIC

Surgical Complications of Peptic Ulcer

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Peptic ulcer, according to all studies, is increasing in frequency in this country, and at present from 2 to 5 per cent of all hospital admissions in general hospitals consist of peptic ulcer patients. It has been estimated that among chronic diseases, peptic ulcer in this country ranks tenth as a cause of death and twelfth as a cause of invalidism and absence from work.

A principle to which most of us can subscribe, I think, is that the majority of peptic ulcers can be handled successfully on a medical, nonsurgical regimen, and perhaps 75 per cent of ulcers in general can be controlled satisfactorily by dietary and medical measures.

It is the complications which call for surgical treatment, of course, and these complications fall into fairly definite categories. Before dealing with the complications which are of surgical significance, however, I would like to speak of a trend which I note among my fellow surgeons and in the literature, namely, the tendency to look with growing suspicion on gastric ulcer as compared with duodenal ulcer.

There is a very high percentage of error in the recognition of the benign chronic gastric ulcer as compared with the early carcinomatous ulcer. In my own series of cases I find that 12 per cent of the patients on whom I have done gastric resection for supposedly benign ulcers turn out to have carcinoma in ulcers. Since the risk of an adequate gastric resection is in terms of 1 or 2 per cent and since the chance of any particular ulceration in the stomach being malignant is considerable, perhaps 10 or 15 per cent, obviously a case can be made out for the primary treatment of chronic gastric ulcers by gastric resection. That is a view that I subscribe to wholeheartedly.

The picture of carcinoma of the stomach, as you know, is an extremely dismal one, and the patients with carcinoma of the stomach that the surgeon is most likely to cure are these early cases in which there is microscopic cancer in the ulcer, sometimes not detectable by any of the diagnostic measures short of serial section by the pathologist. Therefore, a statement which we hear at times now is that chronic duodenal ulcer is a medical disease until complications have arisen which do not respond to medical treatment; chronic gastric ulcer, on the other hand, is a surgical lesion.

In studying the record of gastric surgery for the past five years in our hands with respect to the indications for operation, I find that these indications are about as follows: (1) Perforation called for surgical operation in 24 per cent of this series of cases. (2) Intractability, which has meant for the most part penetration and fixation to the surrounding tissues, called for operation in 42 per cent of the cases. (3) Obstruction of the pylorus or duodenum required operation in 12 per cent, and gross massive hemorrhage necessitated operation in 22 per cent. However, in 40 per cent of this group of cases, two or more of these complications existed in the same patient at the time of operation.

I should like to focus my attention on two principal ones, namely, obstruction and gross hemorrhage. These two complications can be illustrated by the two convalescent patients who are here.

Before going on to the subject of obstruction as a cause for surgical treatment, I would like to speak for a moment of the question of intract-

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ability or penetration. Chronic ulcers of the stomach or duodenum may, of course, burrow into neighboring structures; they may attain enormous size, and they pose some of the most difficult problems in surgery. The intractability when you see the ulcer is on an obvious basis. No longer is the disease confined to the stomach, in many instances, but the pancreas, the liver, the parietal peritoneum, or other structures may be involved; therefore, the gastroenterologist has been treating not ulcer alone but inflammatory disease of other intra-abdominal structures, and it is not surprising that the success of nonsurgical management, in this type of case, is limited.

Figure 1 is an x-ray of the stomach of a 74-year-old man with a high lesser curvature ulcer which I think is well shown. This lesion had burrowed into the pancreas. The patient was suffering from constant severe pain in the abdomen and in his back, vomiting, with severe depletion, hypoproteinemia, and anemia. It was necessary to treat this by total gastrectomy, which in this instance was done through the chest by a transdiaphragmatic approach.

Another burrowing ulcer of the same type is shown in Figure 2; this patient is a 49-year-old woman. The ulceration is enormous. The base of the ulcer was the pancreas, and in treating that case it was necessary to do a total gastrectomy and to remove the spleen and part of the pancreas. It seems to me that one could never hope to treat successfully a large, fused inflammatory mass in this type of lesion, which is unusual, of course, by dietary measures.

The specimen after operation may be seen in Figure 3.

Another illustration of a penetrating, burrowing type of ulcer is shown in Figure 4. In this instance a gastrocolic fistula resulted and the barium you see in the stomach was given by means of a barium enema. This patient was treated with a two-stage operation, a preliminary proximal colostomy, and later resection of the stomach and colon.

When we come to the matter of obstruction from chronic gastric or duodenal ulcer, there are in general two varieties. Obstruction of the pylorus or duodenum can result from active lesions in the vicinity producing edema and spasm. On the other hand, the obstruction may be due to chronic cicatricial stenosis with or without superimposed spasm. In many instances there may be obstruction of the pylorus when the ulcer lies rather remotely from the pylorus, presumably a matter of spasm. Those cases can be handled much more successfully by the standard method of naso-gastric suction, attending to fluid requirements, and maintaining nutrition, than can the cases that have cicatricial organic block in the region of the pylorus. Unfortunately, however, it is frequently impossible to make this distinction on radiographic study. One does not always know whether the obstruction present will respond to nonsurgical treatment. These cases are very interesting ones and call for very nice judgment in their management.

The clinical picture usually is that of persistent vomiting with or without pain; dehydration may develop rather rapidly; there may be hemoconcentration with red cell count elevated and hematocrit elevated; there may be even oliguria and elevation of the nonprotein nitrogen. When the dehydration is corrected, however, we may find that actually an anemia exists, or a hypoproteinemia. The stomach is ballooned out; it may be atonic; its wall may be edematous—very poor tissue for surgical procedure. There may be a secondary gastritis. There may be decaying food present in the stomach. Incidentally, in my experience it is almost

Figure 1. High penetrating lesser curvature benign gastric ulcer; total gastrectomy by transthoracic approach.

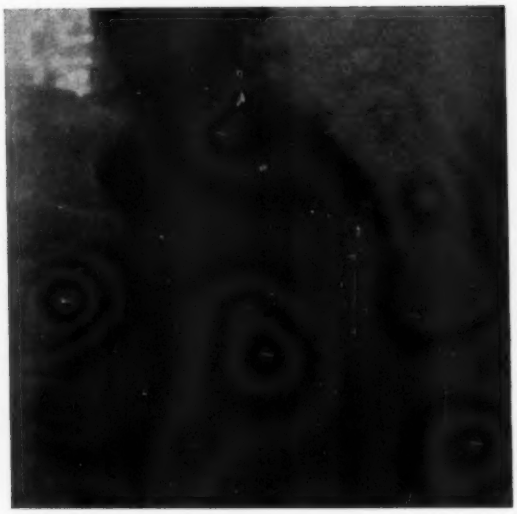

Figure 2. Benign gastric ulcer with penetration of pancreas; total gastrectomy, splenectomy, resection of pancreas.

impossible to empty out one of these hugely dilated stomachs satisfactorily by a naso-gastric suction procedure, or even by the old-style large gastric tube passed by mouth.

A word should be said about the use of x-rays in this situation. Sometimes we are over-enthusiastic regarding these patients with pyloric obstruction, and try to fill the stomach enough to outline it completely, or we persist in an attempt to show the type of lesion at the pylorus, and we may do the patient a good deal of harm by filling up his stomach with barium which is very difficult to remove. I think the x-ray should be used cautiously in the diagnosis of this condition.

THE management which can be recommended in such cases is essentially treatment of the patient's general condition and decompression of the stomach. The general treatment involves fluid and electrolyte replacement; it also involves administration of vitamins and proteins and, in many instances, blood transfusion, these measures being controlled by fairly frequent use of laboratory determinations.

The naso-gastric tube is passed and it is kept on

Figure 3. Benign gastric ulcer with penetration of body of pancreas.

Figure 4. Penetrating benign gastric ulcer and gastrocolic fistula; two-stage repair.

constant suction. It is irrigated at frequent intervals in the hope of favoring better drainage and making certain that the tube is open at all times.

Presently a decision has to be made in this program. Is the patient improving? Will he continue to improve, or has he progressed as much as one can expect? That is a very difficult question. All the improvement may result from the general treatment of the patient while the obstruction in the pylorus or duodenum persists. One can get some help on this question by careful measurement of gastric balance, that is, measuring all the fluid obtained from the stomach and balancing against that fluid which has been taken by mouth or used in irrigation. Actually, however, if after two or three or four days it becomes evident that a high degree of obstruction still persists, and if the patient's general condition has been properly treated, then I think it is time to face the indications for surgical treatment.

Figure 5 illustrates the case of a 52-year-old man with a chronic duodenal ulcer and with gastric tetany and alkalosis, which results where a great deal of acid secretion has been lost through the vomitus, with reduction in the chloride values in the blood. This patient was treated about as I have outlined, and after three or four days was operated upon and subtotal gastric resection was carried out.

Another patient was a 70-year-old man who came into the hospital with a history of vomiting and epigastric pain over a period of about four weeks. During the week before admission to the hospital he said that he had vomited everything he had taken by mouth. The patient was anuric at the time of admission. The nonprotein nitrogen was elevated; his red count and hemoglobin values were elevated, as were his plasma protein concentrations. He had unusually severe dehydration. This x-ray of the stomach was made before the patient came into the hospital, otherwise it would be hard for me to argue that we do not like to put a lot of barium into the stomach in this situation. You can see, however, from the moth-eaten appearance of the gastric wall, particularly in the fundic area, that there is a good deal of retained food and a good deal of gastritis present.

This patient was treated with proper fluids and electrolytes and vitamins as required. A careful measurement of naso-gastric balance was made, and we found that after three or four days the evidence indicated that he still had complete block of the pylorus; therefore he was operated on. In this instance a posterior gastroenterostomy was

done with a jejunostomy for continued feeding and maintenance of fluid balance.

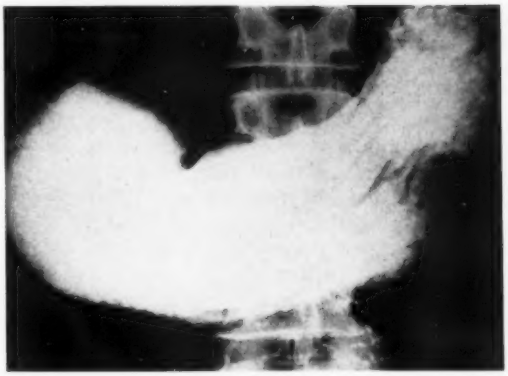

Figure 5. Chronic duodenal ulcer with cicatricial stenosis, gastric tetany, gastritis, and anuria from dehydration; restoration of fluids and electrolytes followed by posterior gastroenterostomy and jejunostomy for alimentation.

DISCUSSION OF PATIENTS

The first patient is presented through the courtesy of Dr. Ernest Bright. The brief abstract of the case illustrates pyloric stenosis due to duodenal ulcer, successfully treated surgically.

This man is 60 years old and was admitted to the hospital May 29, 1948, complaining of pain in the pit of his stomach, and vomiting. He had had recurrent attacks of upper abdominal pain for about five years and recurrent attacks of vomiting for three or four months prior to admission. Pain was described as gnawing in character. He had lost 30 pounds in weight in the three or four months prior to admission to the hospital.

Laboratory values at the time of admission: hemoglobin 17.9 gm. per 100 cc.; red count 6,000,000, indicative of the dehydration previously referred to. On gastric analysis there was free hydrochloric acid ranging from 30 to 106 units. X-ray studies were made, and obstruction, presumably from a duodenal ulcer, was demonstrated.

The patient was placed on gastric suction and given parenteral fluids, and on June 2, 1948, subtotal gastric resection was done. There was a large duodenal ulcer just distal to the pylorus. The ulcer was not resected, but all of the pyloric mucosa was removed. The convalescence was uneventful and he was discharged on the twelfth postoperative day.

I have talked with this man. At present he is in very good health. He has no restrictions in his diet. He has regained 30 pounds in weight. He is back at work. In fact, he has made a complete recovery in every way.

The second complication which I want to dispose of very briefly is that of acute massive hemorrhage from peptic ulcer. The second patient illustrates this complication and its successful management. A brief abstract of his case is presented through the courtesy of Dr. McGinnis.

This man is a 55-year-old clerical worker who came into the hospital January 28, 1948, because of diarrhea, with 15 pounds weight loss, and tarry stools during the previous three or four weeks. He had had a previous admission to the hospital March 2, 1944, because of several bloody and tarry stools associated with weakness and faintness. His blood pressure when first seen, March 1944, was 78/55. He had given a rather vague history of abdominal symptoms for the previous two years. One year before, an x-ray of his stomach and duodenum was said to have been negative.

On admission the hemoglobin values were 8.2 gm. per 100 cc., the red cell count 2.79 million. These values subsequently fell. His red count on the day before operation was 1.64 million. He received 1500 cc. of whole blood within the next three days, but his pressure continued to remain below normal and he continued to pass tarry stools. Therefore, on the fifth hospital day subtotal gastric resection was performed. A chronic thickened duodenal ulcer was found on the posterior wall and two bleeding points were visible at the base of the ulcer. The ulcer was not resected, but subtotal gastric resection was performed, during which procedure the patient received an additional 1700 cc. of whole blood. The postoperative course was uneventful and the patient was discharged on the fifteenth postoperative day.

The case just described illustrates, therefore, the management of massive hemorrhage from a duodenal ulcer by subtotal gastric resection during the phase of acute hemorrhage and, very importantly, with adequate blood replacement before and during the operation.

The patient got along reasonably well following that operation in 1944, until the second admis-

sion. At that time he came in with a history of epigastric distress and vomiting and difficulty with his bowels—diarrhea. His laboratory studies showed severe anemia and depletion, elevation of the nonprotein nitrogen, and a reduction of the carbon dioxide-combining power of the plasma to 49 volumes per cent. The barium enema showed the barium to enter freely into the stomach. In other words, this patient had developed, during the three and a half or four years, a gastrocolic fistula on the basis of a marginal ulcer.

On the ninth hospital day a gastrojejunocolic fistula was resected and end-to-side retrocolic gastroenterostomy was done. The patient's convalescence was interrupted by evidence of a subdiaphragmatic abscess, but subsequently his progress has been extremely satisfactory.

I have talked with him and he is a very grateful patient. He takes three meals a day now almost without restriction and he has regained his weight. There are no symptoms referable to his stomach.

I would like to add a word about this method of handling acute massive hemorrhage from peptic ulcer. There is a growing interest in the surgical treatment of massively bleeding gastric or duodenal ulcer following adequate blood replacement. At present we are running a series of cases using, roughly speaking, alternate cases as controls. We operate on about half the patients, and about half of them we treat by blood transfusions and other medical measures. The result tentatively supports the conclusion that these patients can be operated on successfully by subtotal gastric resection after adequate blood replacement.

The mortality rate in 40 patients operated upon has been 12.5 per cent; the mortality rate in those not operated upon being 25 per cent. All the patients were bled out at the time operation was undertaken.

VAGOTOMY MORE SUCCESSFUL COMBINED WITH GASTROENTEROSTOMY

Gastroenterostomy performed concurrently with vagotomy has yielded encouraging results as a treatment for duodenal ulcer, five Duke University physicians report after a four-year study of the effects of vagotomy in 104 patients with intractable, frequently recurring, or complicated peptic ulcer.

The studies indicate increasing belief that the usefulness of vagotomy is limited by side effects or complications rather than by failure of ulcers to heal. Vagotomy has effected ulcer healing after gastroenterostomy or subtotal gastric resection, and has aided treatment of benign ulcer, but persistence or recurrence has been frequent.

The conclusion is that gastroenterostomy should be added to vagotomy if possible when this operation is performed in preference to gastric resection. The studies were made by Drs. K. S. Grimson, R. W. Rundles, G. J. Baylin, H. M. Taylor, and E. J. Linberg.

RATING SYSTEM ESTABLISHED FOR MENTAL HOSPITALS

An inspection and rating system for mental hospitals in the United States and Canada has been established by the American Psychiatric Institute for the purpose of stimulating the same kind of voluntary improvement in public and private mental hospitals that a similar procedure developed in 1918 by the American College of Surgeons achieved in general hospitals.

Dr. William C. Menninger, the Association president, has announced the appointment of Dr. Ralph M. Chambers, until recently superintendent of Taunton State Hospital, Taunton, Massachusetts, as director of the project. A central inspection board of ten psychiatrists under the chairmanship of Dr. M. A. Tarumianz, superintendent of Delaware State Hospital, will serve as the governing body. The work is made possible by a grant from the Psychiatric Foundation.

The plan is for the hospitals to request inspection (many already have done this). The institutions will be appraised against a set of minimum standards covering every phase of operation and facilities and recommendations made after inspection. If improvements do not bring an institution up to standard within a reasonable time, the institution will be rated as sub-standard. This process will provide administrators, boards, legislative, and community leaders with a factual basis for their efforts to obtain appropriations or other measures needed to achieve the desired improvement.

DIAGNOSTIC CLINIC

Diet in the Treatment of Hypertensive Disease

IRVINE H. PAGE*, A. C. CORCORAN†, AND ROBERT D. TAYLOR†

CLEVELAND CLINIC, CLEVELAND

THE quickest way to start an argument among the people who work with hypertension is to mention the Kempner rice diet, a low sodium diet, or any kind of diet at all. That is for a very good reason. The trouble is that the disease in the patients in whom you are studying its effects has protean manifestations.

Hypertension, as you know, runs the gamut from a very malignant form to forms that are extremely benign, and that is what causes the confusion. Another factor contributing to the confusion is that there are not enough objective methods used in the study of the patient. As you all know, this business of taking a few blood pressures, calling that the control period, and starting the diet or treatment of the patient leads to confusion. If you will analyze most of the papers on the subject you will find that the difficulty usually comes from the fact that the cases have not had a sufficiently long control period. (Figures 1 and 2.)

The blood pressure alone, in short, is an insufficient datum on which to follow the course of the disease. One must follow other changes, especially those in the arterioles of the eyegrounds, before one knows what he is doing. If the blood pressure is accepted without sufficient control, you may very easily be misled. That, I think, is probably the chief cause for misunderstanding in the use of diet. If you will think over your own cases, you will recall that many of the most dramatic falls in blood pressure occurred in the absence of any definitive treatment and so reflect the variability of the disease.

There is still one other point that I think should be mentioned. It is becoming increasingly apparent that hypertension is no simple disease. We like to catalog it and say that it is renal; it is this, that, or the other; in other words, we like to think there is some primary, easily discoverable cause. Yet the good old days are over when we could tell exactly the cause of hypertension. The more we study it, the more we realize that it is probably of multiple etiology, and it is the constant interplay, the mosaic, which finally spells the entire clinical picture of hypertension.

There are some patients in whom the nervous factors predominate, and one might almost think the nervous system was the primary cause of the disease. It may well be. There are others in whom it is perfectly obvious that the renal factors are the predominant ones. There are others in whom the adrenal factors are most important. It is very likely that in this whole hodge-podge of disease we have cases in whom the etiology is not the same. That is important, because it is quite clear now that some patients respond to dietary treatment and many do not, so one cannot say categorically that hypertensives respond or do not respond; it depends entirely with what you are dealing.

My task is really to set the problem, and Dr. Corcoran will discuss briefly the physiologic factors and Dr. Taylor will present the clinical phase of our own results.

Let me review for you the different types of dietary treatment which have been really seriously

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considered as treatments for hypertension. There is, first, the low calorie diet with weight reduction. I think everybody recognizes that weight reduction reduces the amount of tissue through which the heart has to pump blood and therefore it is an accepted way of treating hypertension; I believe there is no argument about that. The war experience has been of a good deal of interest in this connection. As you know, we got reports from Europe that in almost all cases the blood pressure went down in people who had been in concentration camps and had undergone great deprivation and starvation. Indeed, the blood pressure of the entire population went down, and on feeding these people it began to go up again. I think that almost proves that a low calorie diet is beneficial as regards hypertension.

Now we go to a little more specialized form of diet. There is an old belief that protein in some way or another is disadvantageous to the patient with hypertension. That is an idea which at the present time has been largely discredited. The low protein diet is used hardly at all. Yet one is not sure whether it should have been discarded as quickly as it was. It might possibly be that there are amino-acid deficiencies that result from low protein diets which have a more specific effect on arterial pressure than does an overall low protein consumption.

The third form of diet is the low salt diet. That is the one that is being most actively discussed, and, as you know, many people believe that any effects of the rice diet, the so-called Kempner diet, are due chiefly to the fact that it is a low salt diet.

As you all know, it has taken almost half a century to reconnect the kidney with the circulation, thanks to undue preoccupation with the cardiac function of the vascular tree. Back in 1903, Widal was probably the first to point out that edematous nephritics excrete little salt and water. In 1932, Newburgh showed that if adequate water was given there was normal excretion of salt and water. Achard in 1901 showed that salt given in congestive failure is not readily excreted and that edema increases. Schemm in 1942 made his important contribution that cardiac edema can be effectively treated without fluid restriction if sodium is restricted, and Schroeder showed the importance of sodium versus chloride.

Ambard in 1905 recommended chloride restriction for treatment of hypertensives, but he had no very good reason for doing it and because the studies were not very adequate it was dropped again. Allen in this country and Volhard in Germany recommended the diet again, and in going over Allen's results I think one must admit that some of the cases did show definite falls in blood pressure, but again it was a very controversial issue and after several other studies had failed to confirm these results, the method was largely dropped again.

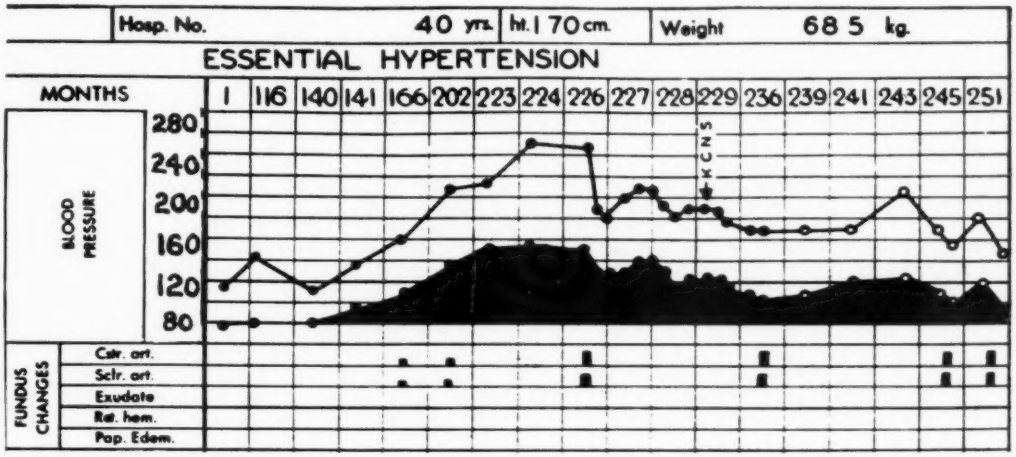

Figure 1. Each dot represents the average of 14 blood pressure readings, and as shown, there have been fluctuations in the blood pressure. Fluctuations may be so great as to make it impossible in some patients fairly to evaluate an experimental treatment. Compare with Figure 2.

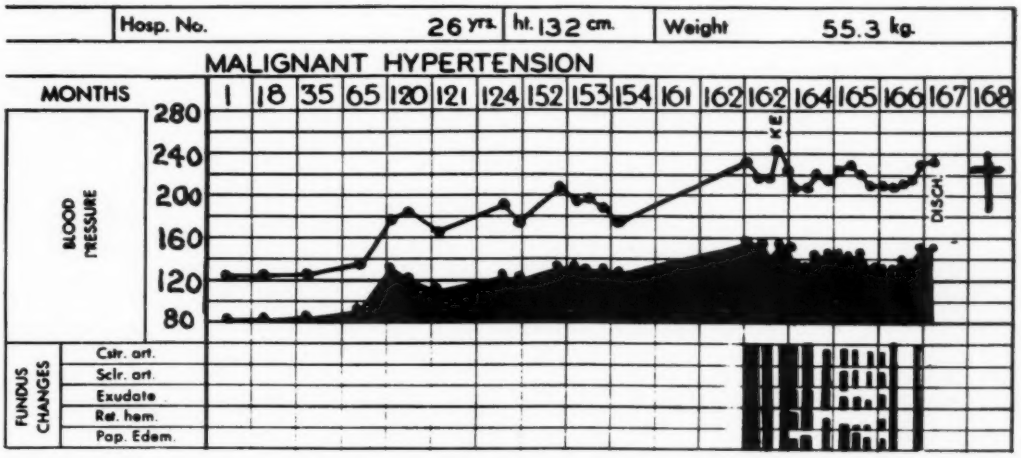

Figure 2. The record here is similar to that shown in Figure 1. The blood pressure steadily increased and the patient died at the end of the study.

The importance of salt in the production of vascular lesions by desoxycorticosterone acetate was shown by Selye. Grollman and Harrison in 1945 had 6 cases which were reasonably well studied and 4 did show definite falls in pressure. A Hollander named Viersma, whose work I came across but recently, did a beautiful but limited study on this subject. No overall adequate study has subsequently appeared, and that is where the matter now stands. Like many other clinics, we have been actively and belatedly studying the matter for the past three years.

I am sure you are all familiar with the Kempner diet. It is a very boring one. Many patients will not stay on it. On the other hand, there is little doubt that some patients seem benefited if they will continue the diet. We believe that it is to be thought of as an experiment well worth prosecuting but not as an established therapeutic procedure. I am sure Dr. Kempner would agree.

There is one other diet which is also highly controversial, and that is the low cholesterol diet in the treatment of vascular diseases. As you know, there is a definite belief that the ingestion of cholesterol leads to the deposition of cholesterol in the vessels and that, of course, is particularly important in hypertension. Views on the usefulness of this diet, especially in coronary atherosclerosis, are also highly variant. I recently attended a meeting in Chicago of the American Society for the Study of Arteriosclerosis where about every other speaker wavered one way or the other.

I think one could say categorically that the evidence is wholly insufficient at the present time to withdraw cholesterol-containing foods from the diet. If there is manifest hyperlipemia, that is another proposition, because I think that if hyperlipemia is long sustained, it unquestionably leads to the deposition of cholesterol in the vessels. It may well be that a low cholesterol diet is advisable, but in the management of patients with hypertension or with coronary disease I think there should be much better evidence than is currently available to justify withdrawing important food elements such as milk, butter, etc., from the diet on the basis that one was going to prevent the further spread of arteriosclerosis.

To review briefly, the diets said to be useful in the management of hypertension are: (1) the low calorie diet, a successful form of treatment; (2) the low protein diet which should be restudied, I think, and any of you who want to take up an important clinical problem will be doing the field a great service if you will tackle that one; (3) the low salt diet, and the variation between the ordinary low salt diet and the drastic reduction, that is, getting the sodium below 200 mg. in the urine; (4) the Kempner rice diet, and (5) low cholesterol diet.

PHYSIOLOGIC FACTORS

In considering the physiologic indications for the various forms of dietotherapy, let us go over them more or less in the same order as Dr. Page has done.

First, as to the low caloric diets, what are they known to do? What could be their usefulness? Weight reduction is known to decrease cardiac work. While the estimates vary, one accepted figure is that a 3 per cent decrease in body weight leads to a 10 per cent decrease in cardiac work. Such a decrease in cardiac work can and must be of advantage to the hypertensive patient and more particularly to the patient whose cardiac function is in some degree prejudiced. So we have a good solid basis for a low caloric diet, in some people at least, certainly in those who are overweight.

What about low protein diets? The one definite indication that is established beyond any doubt is that a low protein diet does decrease azotemia in patients who are in renal failure. Such diets greatly increase survival in nephrectomized animals. Very recently there has been reported the successful treatment of terminal renal disease with diets containing approximately 0.5 gram of protein per kilogram of body weight. Such diets have a real indication and a real field of usefulness, more particularly when the total calories are more than are usually considered adequate.

Then there is the experimental indication for a low protein diet in hypertensive disease. Selye has given animals a crude mixture of anterior pituitary powder. Animals given this powder sometimes develop hypertension and arterial disease. Hypertension and arterial disease occur more readily and more extensively when the diet is rich in protein and more particularly when it is rich in certain proteins, as, for instance, casein; it does not occur when the diet contains lactalbumin. That is an experimental indication which it is difficult at this point to analyze. The suggestion, of course, is that in patients with Cushing's disease, for example, a low protein diet should be recommended rather than the high protein diets which are customarily used, since such a low protein diet may decrease the production of adrenotropic hormone and cut the vicious cycle of this unusual disease. But the carry-over to clinical essential hypertension is not at all clear.

What are the definitive indications for the low sodium diets? Of course, glomerulonephritis with

edema, congestive heart failure, cirrhosis with ascites, and other conditions in which sodium is abnormally retained may be listed. An experimental indication at which Dr. Page hinted is again derived from the work of Selye. Rats treated with desoxycorticosterone in huge doses develop arterial disease and hypertension. When sodium is removed from the diet that arterial disease does not develop. When sodium is added to the diet the disease becomes much more severe, and when either the potassium or ammonium ion is substituted for sodium, the arterial damage is minimal. There is an experimental indication for sodium restriction in conditions associated with nephrosclerosis or at least in those in which adrenal hyperactivity is suspected.

Sodium retention in heart failure is of special interest. The mechanism of the sodium retention in congestive heart failure is not fully established. It may in part be a combination of adrenal activity with an increase in the production of steroids of the desoxycorticosterone series and a positive sodium retention. There is also another mechanism. In the patient with congestive heart failure, especially during even mild exercise—as when he is walking around—the renal blood flow falls off very seriously and as the renal blood flow and glomerular filtration fall, the amount of salt which can be excreted decreases and the patient tends to retain sodium as a result of his daily activity. Treatment is bed rest, low sodium diet, adequate fluid, and digitalis and mercurials.

With the regard to diet and vascular disease in the average patient, we have the indication in overweight for a low caloric diet; in azotemia and renal failure, for a low protein diet; and of a low sodium diet in cases of sodium retention in congestive heart failure, in Cushing's disease, and, possibly, as Dr. Page has mentioned, in hypertension.

The evidence, both experimental and clinical, in hypertension itself is much more tenuous and there is really no good sound physiologic ground for saying that this or that patient is going to respond to a low sodium diet by a reduction of blood pressure; even when a reduction of blood pressure occurs we do not know the mechanism. It is probably not a failing of adrenal activity. Such evidence as we have seems to indicate a deviation of the activity of the adrenal cortex into some other line of steroid formation from those which maintain hypertension.

We have these positive indications. All of them are to a certain extent met with the rice diet. It is low in sodium, low in calories, rich in carbohydrate, low in protein. It is, in a word—and in a good sense of that word—a shotgun prescription. But the indications for sodium restriction alone are better met with a low sodium diet. Neither the rice nor the low sodium diets are very palatable. I refer you to Job VI:6, "Can that which is unsavory be eaten without salt?" as a suitable text to this point.

As to the physiologic effects of these diets, we should remember that the low protein and low caloric diets and those low in sodium tend to decrease blood and interstitial fluid volume, and that this effect may be a very desirable one in some patients. In most, the consequent decrease in the over-all renal excretory power which occurs is not a serious matter at all. They are not taking very much, they are not putting out very much. A little decrease of kidney function due to a decrease in blood volume is not a bad thing. However, in a patient whose renal function is already prejudiced, whose kidneys cannot do their normal work of maintaining in the body normal concentrations of sodium and which cannot respond to a decrease in sodium by grabbing onto the sodium in tubular fluid and transferring it back into the blood, a low sodium diet may be perilous. The result of sodium depletion is a state of renal failure and azotemia, extreme weakness, a sort of Addison-like syndrome.

If a patient happens to be on a low sodium diet and is subject to vomiting, diarrhea, or severe trauma, with consequent sodium loss, the need for careful control is imperative, even in the absence of any severe renal failure.

CLINICAL STUDIES

I AM to report some of the results we have observed among patients with hypertensive vascular disease receiving low sodium diets. To digress a moment, it seems a sad commentary upon American medicine that two of our present vogues are in reality pretty ancient. I think it was around 1905 that the Mayos suggested the use of vagotomy in the treatment of peptic ulcer. It was

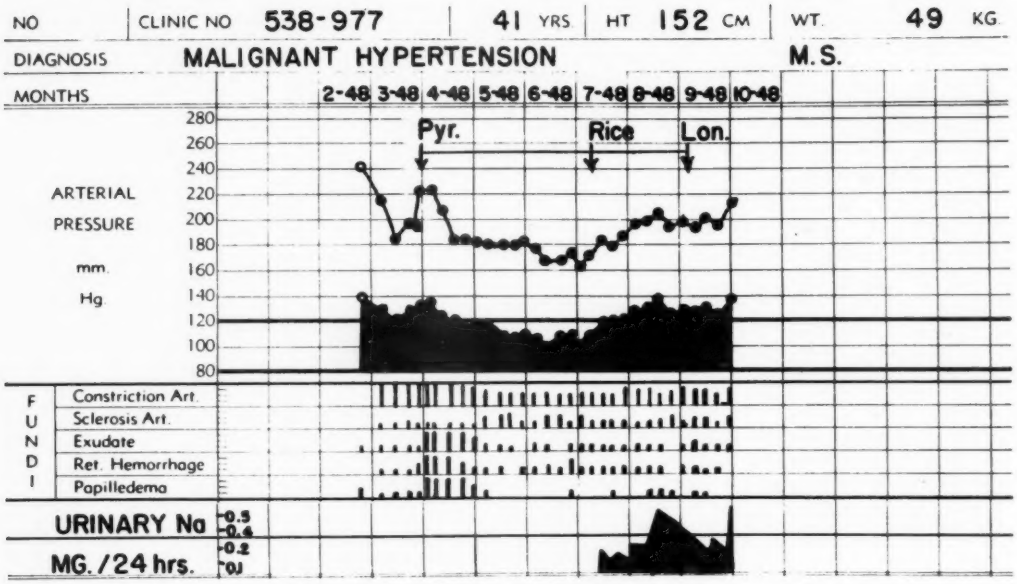

Figure 1.

kicked around at that time and then put on the shelf until the early part of 1940 when it was revived. At the present time it is a very useful means of arresting the progress of this disease. As Dr. Page has indicated to you, low sodium diets were first suggested around the turn of the century. It is difficult to know why we as medical people are so slow to recognize such useful procedures.

We have learned several things from the treatment of patients with low sodium diet. First of all it is extremely difficult to accomplish adequate restriction of sodium. Drs. Landowne and Alving of Chicago found it almost impossible to give an effective low-sodium diet to an ambulatory patient. Sodium, like bacteria and the poor, seems to be omnipresent. Further, it is very difficult to give a patient an edible diet that he can follow while carrying on normal activities, and yet restrict his intake of sodium to a degree sufficient to have any effect upon his blood pressure. Consequently, those patients we have studied have been in the hospital. Their intake of food has been carefully regulated by competent dietitians, so that it contains less than 200 mg. of sodium per twenty-four hours. As a further check we have measured the output of urinary sodium over twenty-four-hour periods. Even honest and cooperative patients in good hospitals will come up on some days with a gram or two of sodium and the Lord Himself will not know whence it came. It may have been in toothpaste, mouth wash, drugs, or drinking water. We are quite convinced that it is difficult to attain a diet that will restrict sodium adequately.

In addition to these problems, there are relatively few patients who respond favorably as far as blood pressure is concerned. Probably at this time we are not justified in making a percentage estimate, but in the patients we have treated, who number some 50, less than 20 per cent have experienced a reduction of blood pressure or other objective improvement following such a program.

Too frequently the course is not unlike that of a patient whose course is illustrated in Figure 1. This patient had been treated previously with some experimental materials with which we have worked for some time. As you see, the blood pressure, systolic and diastolic, had dropped from control levels of 220/130 down to an average of 170/105. The retinopathy as represented by papilledema, hemorrhages, and exudate, had regressed until the hemorrhages were but intermittently present. The exudates disappeared. She was doing quite well.

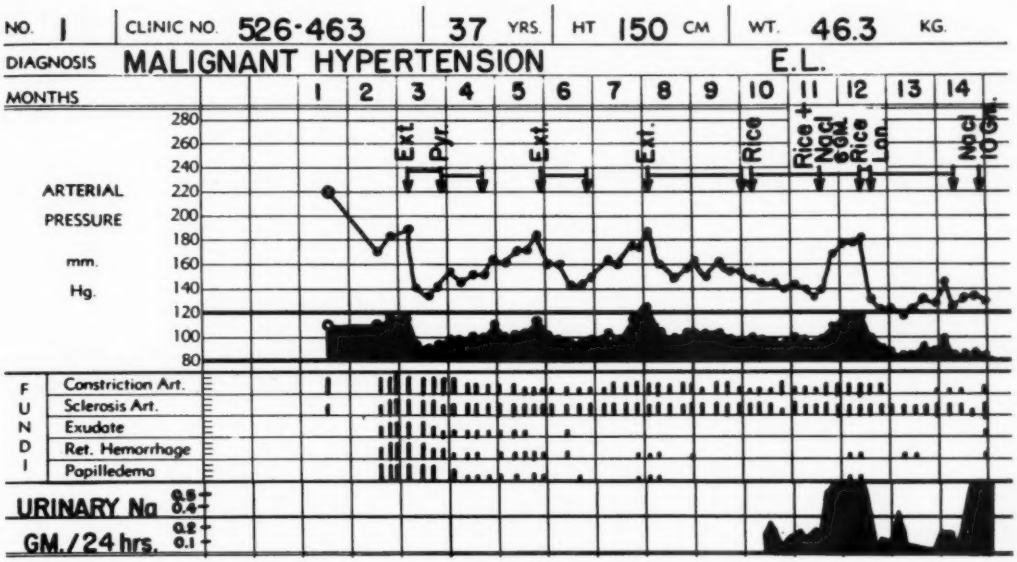

Figure 2.

Previous therapy was discontinued and her intake of sodium was restricted by giving her a diet which Dr. Kempner suggested, known as the "Rice Diet." Her urinary output of sodium was reduced to less than 200 mg. in twenty-four hours. During this time, a total of about two months, her blood pressure rose to and above its previous levels, papilledema returned, retinal hemorrhages were more frequently seen, and her disease progressed merrily.

At this point a diet which Dr. Page devised was instituted. It consists of a milk protein* which is essentially free of sodium and of meats, fruits, and vegetables of low sodium content. On this schedule her urinary sodium continued to be in the neighborhood of 200 mg. per twenty-four hours. The blood pressure continued to rise and she went on to die of a cerebral hemorrhage. All patients fortunately do not respond in this manner. How are we going to select which patient is going to have a good result? We certainly do not know at the present time, except to try.

A rather different course is shown in Figure 2. This patient was a woman 37 years of age. She had been treated with various other materials up to this point. Her blood pressure by these means had been reduced in the tenth month of her disease to an average of 140/95 to 150/100. Then we thought we would see what would happen to her as compared to the previous case. She was put on Dr. Page's diet, and instead of her blood pressure going up it continued to go down until it reached a level below 140 systolic. Next she was given a rice diet for a period of three weeks and the blood pressure fell further.

While the patient was eating rice, we added in tablet form 6 gm. of sodium chloride per twenty-four hours. Her blood pressure rose to 180 systolic and 120 diastolic. The sodium was discontinued, the rice diet was continued, and the blood pressure came on down. She soon could not tolerate the rice diet and was put on the Lonolac diet. Her blood pressure continued to stay down and her retinal disease was absent. We now gave her 10 gm. of sodium chloride a day; her urinary sodium came up, and for a period of three weeks no change in the blood pressure occurred. However, at present her pressure has started upwards.

*Prepared by Mead, Johnson & Company.

Figure 3 illustrates a case of a woman who had an essential type of hypertension. The blood pressure varied rather widely over a period of a

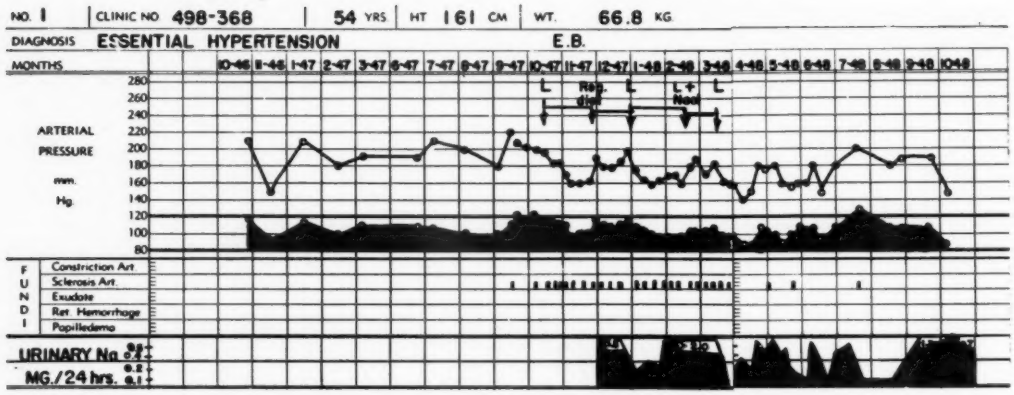

Figure 3.

year before she was treated. It occasionally was 150/90, but usually was up at hypertensive levels. The open dots represent outpatient measurement; the closed ones were taken in the hospital. She was given the low sodium diet, and her blood pressure was reduced to average 160/100. With the restitution of normal diet it promptly came up. The low sodium diet again was started—the blood pressure came down; with sodium chloride added it rose.

As you see, the level of urinary sodium parallels rather closely the height of her blood pressure. When the twenty-four-hour output of sodium is down her blood pressure is down.

Another patient had a favorable result following restriction of sodium. The blood pressure had averaged 180/120. It was reduced to 150/100.

In summary it seems safe to say that less than 20 per cent of patients with hypertension respond to diet low in sodium. Possibly more may do so if the diet is continued for years rather than months but as yet no one has studied such patients. It is very difficult to restrict adequately dietary sodium in ambulatory patients. At present there is no method short of trial which will tell which patient will respond to dietary measures.

YALE CLINIC SETS UP CONSULTANT SERVICE FOR INDUSTRY

A new consultant service to assist industry with the problems of alcoholism among workers has been set up by the Yale Plan Clinic, New Haven, Connecticut. All branches of the clinic, together with the National Committee for Education on Alcoholism, will cooperate in the consultant service, which this year will be limited to the area of the eastern states.

The major aim of the service will be to train specialists in industrial health, employees in personnel and employment departments, and company personnel dealing with discipline to a recognition of the problems of alcoholism and ways to control it.

Dr. George Lolli, medical director, and Raymond McCarthy, executive director of the clinic, are in charge of the industrial consultation, training and rehabilitation services.

CASE REPORT

Diagnosis and Treatment of Cystosarcoma Phyllodes

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THE METAMORPHOSIS of a fibroadenoma of the breast into a tumor which may attain gigantic proportions is rare. This paper is a brief discussion of these giant tumors, and includes what is believed to be the one hundred and twenty-sixth case report of this unusual and confusing clinical entity. The confusion may be more thoroughly appreciated when one is made aware of the fact—brought out by Owens and Adams[1] in 1941—that thirty different names had been employed in describing the 121 examples which had been reported up to that time.

A giant tumor of the breast was first described by Chelius[2] in 1828 as a "cystic hydatid." Ten years later, Johannes Muller[3] gave a detailed description under the name of "cystosarcoma phyllodes," which is still the term most commonly used to designate the lesion.

In order to understand cystosarcoma phyllodes, it is necessary that one first have a knowledge of the benign fibroadenomas of the breast. According to Geschickter[4] fibroadenomas are the most common benign tumors of the breast in young women in the childbearing period. He feels that benign cystic degeneration is not uncommon, but that malignant changes are rare. It has been shown by Oliver and Major[5] that the age incidence in benign fibroadenomas was highest between 20 and 24 years in white women, but that it was almost five years earlier for colored women. Geschickter[6] has experimentally produced fibroadenomas in the breasts of rats by providing a constant, intense estrogenic stimulation. We now know that there is a low estrogen content present in girls prior to the onset of menses, which becomes cyclic or variable during their menstrual life. There are, however, occasions when this estrogen factor increases and remains at that higher level, namely, during early adolescence, during pregnancy, and during the onset of the menopause. At the latter period this increase seems to appear as a final, intense stimulation before dying down. It has been observed clinically that fibroadenomas of the breast tend to occur, or, if already formed, to increase markedly in size, during these three periods. It is in this rapid growth of a preexisting lesion occurring at the menopause that we are particularly interested. In some instances metaplasia may take place and sarcoma changes ensue.

Cystosarcoma phyllodes is a giant tumor of the breast thought to arise from a preexisting fibroadenoma, and is usually seen about the

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time of the menopause. The average age when these occur, according to Lee and Pack,[7] is at about 45½ years. The lesion is most often seen in women who have given birth to several children, but it may occur in unmarried females or even in males.

The history frequently reveals that a small, hard, discrete tumor of the breast had been present for several years, and that it rapidly increased in size. Pain is not present as a rule. The only complaint is apt to be discomfort arising from the immense proportions and dragging weight of the breast. Good health is maintained and constitutional symptoms are minimal.

Examination of the breast reveals it to be tremendously enlarged, firm, and lobular. Usually neither the nipple nor the underlying skin are invaded. Palpation of axillary lymph nodes may be difficult or impossible, for as a rule they are not primarily involved. Microscopically, the predominating tissue is composed of myxomatous stroma, with an interlacing meshwork of star-shaped cells and fibers.

In 1931 Lee and Pack[7] reviewed 105 such cases reported in the literature, adding 4 new ones of their own. Owens and Adams[1] reviewed the literature from 1931 to 1941 and presented 12 collected cases to which they added one of their own. To this group of 122 cases reported in the literature, Cooper and Ackerman[8] reported 3 more in 1943, making a total of 125 cases.

CASE REPORT

A 40-year-old colored female was hospitalized on September 24, 1939, for a tremendous enlargement of the left breast, which had become so big and heavy that it was necessary for her to wear a sling around her neck to aid in the support of this tremendous breast. It was primarily the fact that the sling had rubbed a raw area on the back of her neck that made the patient seek medical attention rather than enlargement of the breast.

The patient stated that she had noticed a lump in her breast eleven years prior to admission. At no time had there been any pain in the breast. When originally noticed, the mass in the breast was described as about the size of an orange. It remained about the same size until two years prior to admission, when it began to get progressively larger. The patient repeatedly stated that her only complaint was the tender sore area on the back of her neck and that the breast at no time caused her any pain. There was a history of dyspnea on exertion. Changes in the menstrual cycle had taken place, indicating the onset of the menopause. The patient had had four children whose ages ranged from 12 to 20 years.

The physical examination revealed her to be moderately obese, and apparently in excellent health, aside from the tremendous enlargement of the left breast and the ulceration on the back of her neck. The blood pressure was 230/138, and it was noted that a rather soft blow could be heard along the left border

of the sternum. Examination of the breast on the right side revealed no nodules nor tenderness, nor was there any evidence of pathologic changes.

Examination of the left breast revealed it to be 30 by 15 by 20 cm. and 75 cm. in circumference. The skin appeared tight and shiny. The areola and nipple were stretched into a flat sheet, but without retraction. The skin did not seem to be attached to the underlying tumor. On palpation there was an irregular nodular structure which had the feel of a bag of oranges. The lobulated masses were moderately firm. No glands were palpated in the axilla. A simple mastectomy was performed on September 27, 1939. The specimen weighed 7,830 gm. (17.2 pounds). The wound healed nicely, and her course in the hospital was uneventful. Figure 1 shows a preoperative picture of the patient, in which the tremendous enlargement of the breast is easily seen.

Figures 2 and 3 are low- and high-power photomicrographs taken from the specimen. Microscopically, the tumor is composed of a large amount of myxomatous tissue, with widely separated cells. The cells are fusiform and stellate, the long streams of eosinophilic cytoplasm is some cases being continuous with that of an adjacent cell. The nuclei are fusiform for the most part, with a finely granular nucleoplasm and no apparent nucleolus. No mitotic figures are seen in these pictures. There is a heavier concentration of these benign-appearing fibrocytes about the blood vessel, and a few ducts have been incorporated in the tumor. The epithelial element has not been affected by the stromal changes.

Figure 1. Photograph illustrating a tremendous enlargement of the left breast: Cystosarcoma Phyllodes.

During the year following the operation the woman was seen approximately once a month. During the next three years she was visited on several occasions, the last time being October 1943. At this time, four years following operation, she still appeared to be in good health without any evidence of local recurrence or any symptoms of bone or pulmonary involvement. Because of the war further visits to the patient were impossible.

It is interesting to correlate the findings of this individual case in an attempt to bring out the possible influence of estrogen stimulation. The patient was first seen two years after the onset of the rapid growth of the lesion, when she was 40 years old. She had been aware of the lesion since she was 29 years old, or for a period of eleven years. However, she definitely described the growth to be about the size of an orange when she first noticed it. Therefore, it may well have been present for many years in order to have attained such a large size before being noted. Another interesting feature is that when this patient was admitted to the hospital she had a daughter 12 years old, who was born one year before the tumor was noticed by the patient. Therefore, we might consider that this woman had a fibroadenoma of the breast which may have developed sometime during her adolescence and that her four pregnancies

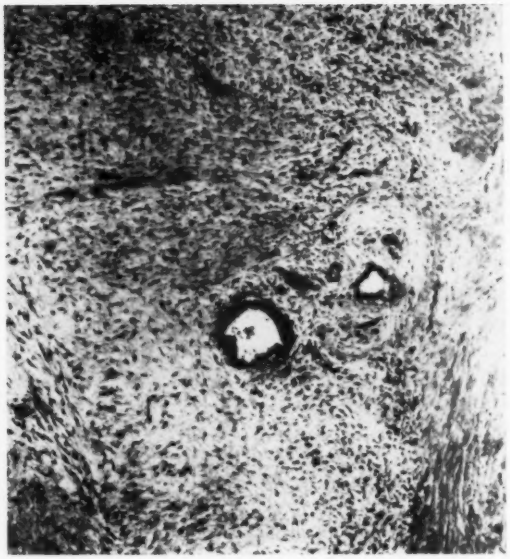

Figure 2. Photomicrograph x 70. This shows the orderly, innocent appearance of the cellular structure. The tissue was taken from the tumor shown in Figure 1: Cystosarcoma Phyllodes.

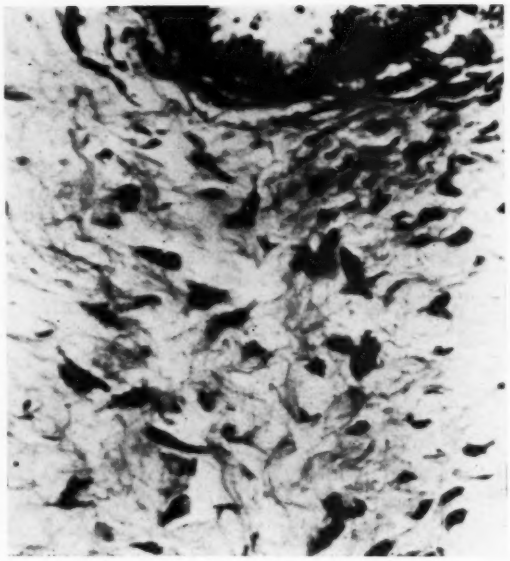

Figure 3. Photomicrograph x 395. An enlargement of a small field of Figure 2 reveals the absence of mitosis, equality of staining, and uniformity of cellular elements: Cystosarcoma Phyllodes.

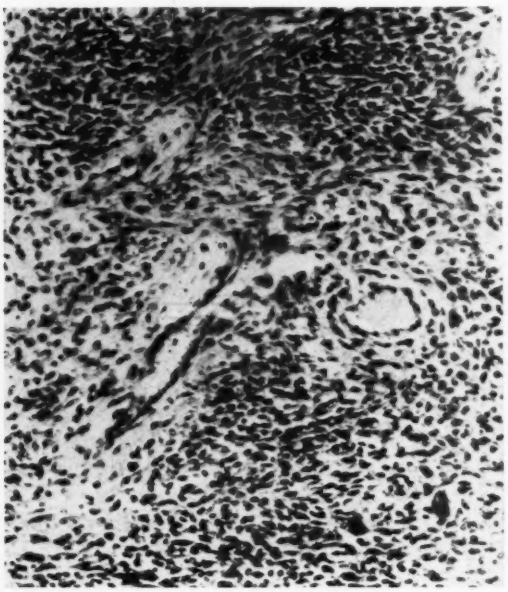

Figure 4. Photomicrograph x 140. This is a section taken from a true sarcoma of the breast for the purpose of contrasting it with Figure 2. A disorderly malignant appearance prevails: Sarcoma of the breast.

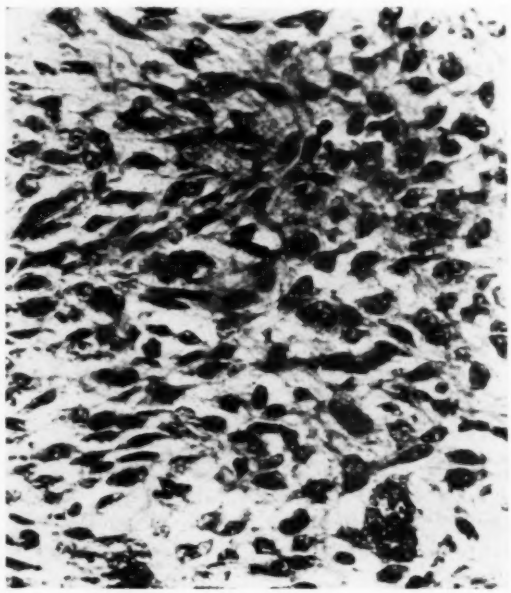

Figure 5. Photomicrograph x 395. This is an enlargement of a small field of Figure 4. Mitotic figures, variability of staining, giant cells, and an overcrowding of cells predominate the picture: Sarcoma of the breast.

Figures 2-5, Courtesy ***A.I.P.***

caused an increase in the size of the lesion until she finally noticed its presence. Later, the increased, constant, estrogen stimulation during the premenopausal period possibly caused the tremendous growth of the lesion.

For the sake of comparison, Figures 4 and 5 represent the low- and high-power photomicrographs taken from the breast of a patient suffering with a true sarcoma. Microscopically, this tissue is variable, with large areas of scattered cells and foci of closely packed cells which lie in a transparent, homogeneous myxomatous material showing a meshwork of fibrous tissue. There is great variation in the size and shape of the cells, some being small and round, others being larger and elongated. Scattered giant cells contain 3 to 5 dark irregular nuclei, and there are numerous mitotic figures throughout. The individual cell contains large irregularly shaped pyknotic nuclei, and one or more small round nucleoli. The cytoplasm, where seen, is eosinophilic, but where the cells are spaced in the myxomatous material, the cell outline either is indistinct or is not visible. It is hoped that when comparing Figures 1 and 2 with Figures 3 and 4 that the benign appearance of this cystosarcoma phyllodes becomes apparent.

About the same time that Lee and Pack[7] had collected 109 cases of cystosarcoma phyllodes, D'Aunoy, Rigney, and Wright[9] reported 514 cases of true sarcoma of the breast collected from the literature. The frequency of occurrence seems to be about 1 cystosarcoma phyllodes for every 5 true sarcomas of the breast. In resuming their findings, they stated that the average age was 44 years. They may occur in males and unmarried females. There is rapid growth, large size, maintenance of relatively good health, and little involvement of lymph nodes. The only difference in the history is that the rapid growth period is measured in weeks for true sarcomas and in months or years for cystosarcoma phyllodes.

Cooper and Ackerman[8] state that cystosarcoma phyllodes, like all neoplasms, varies in potentiality from a very benign to a very malignant type of tumor. It is felt that in general the prognosis is relatively good, because the lesion is usually benign. However, it may undergo metaplasia and even metastasize to the lungs and bones, or may recur locally following its removal. Ewing[10] says that the tumor may perforate the skin with a resultant false appearance of malignancy.

The treatment of choice is simple mastectomy without axillary dissection. Slaughter and Peterson[11] in a recent article on the indications for doing a simple mastectomy specify cystosarcoma phyllodes as one of the lesions to be treated by this definite but limited therapeutic measure. The teaching of Bloodgood[12]—that in general a simple mastectomy is too radical a procedure for a benign breast lesion and yet too inadequate for malignant tumors—is a good rule of thumb. However, the treatment of cystosarcoma phyllodes by simple mastectomy would appear to be a justifiable exception.

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STATISTICAL SURVEY

When Shall Ovaries Be Removed?

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THE purpose of this paper is twofold: (1) To tabulate the incidence of various diseases of the ovary, and (2) to demonstrate the high percentage of ovaries removed having no pathologic lesion.

A summary of the lesions observed in 472 ovaries is shown in Figure 1. By far the commonest disease of this organ is ectopic endometriosis. My criteria for the diagnosis of endometriosis is the presence of both endometrial glands and stroma either on the surface or in the substance of the ovary. Many sections with prolonged study are often required to demonstrate both of these features. Solitary cystomas are simply large follicular type monoloculated cysts having a diameter of greater than four centimeters. Two cases of Meigs' syndrome were encountered in this series, one associated with a fibroma and the second with bilateral serous cystadenocarcinoma.

The first series of ovaries studied were removed during the years 1939 to 1942 inclusive and consisted of 1,181 cases. The operations were performed in five private hospitals and in one county hospital. Four of the private hospitals are located in small towns of less than 25,000 population while the fifth is a large general hospital in a city which has a population of 125,000.

In the large private general hospital series only 28.1 per cent of the ovaries removed showed a pathologic lesion. The four smaller private hospitals were lumped together for statistical purposes and here the percentage of ovaries with lesions was only 29.4 per cent. At the county hospital where there could not possibly be any element of remuneration 54.1 per cent presented a lesion. Figure 2 shows a tabulation of these statistics.

In classifying ovaries with no pathologic lesion the following criteria were used:

(1) All ovaries removed because of primary malignant disease in the cervix, uterus, or fallopian tubes were not counted.

(2) All ovaries from individuals over 60 years of age with no lesion were disregarded.

(3) Follicle and corpus luteum cysts less than 4 cm. in diameter were considered as normal.

(4) Corpora hemorrhagica, lutei, and albicantes are normal physiologic processes and were so regarded.

The commonest preoperative diagnoses in the normal group were appendicitis, cystic ovaries, endometriosis, ovarian hyperfunction, painful ovary, and pelvic inflammatory disease. The commonest postoperative diagnoses were cystic ovaries, oophoritis, and ovarian hyperfunction.

Because of the high percentage of ovaries removed showing no pathologic lesion I decided to attempt to determine the reason for this and then take any steps I could to reverse these statistics. After numerous consultations with the physicians performing the surgery it became apparent that the main reason for the removal of a normal ovary was the failure of the surgeon to be able to distinguish from a gross pathologic standpoint the normal functional physiologic changes in an ovary from true lesions. Many of these men had interpreted simple follicle cysts, corpus luteum cysts, hemorrhagic follicles and corpora lutei seen at the operating table as pathologic lesions. These, of course, are normal physiologic changes which do not justify surgery. They are usually symptomless and only the most conservative operation is justified. The ovary is an organ with complex functions so that even if it is causing trouble in one direction it may be still more

SUMMARY OF PATHOLOGIC LESIONS

Lesion	*No. of Cases*	*Per Cent*
Ectopic endometriosis	132	27.9
Solitary cystoma	96	20.3
Dermoid cyst	49	10.3
Infection and abscess	45	9.5
Multilocular serous cystadenoma	41	8.6
Multilocular pseudomucinous cystadenoma	28	5.9
Cystadenoma with carcinoma	19	4.0
Fibroma	15	3.1
Infarction	11	2.3
Primary carcinoma other than from cystadenoma	9	1.9
Metastatic carcinoma to ovary	6	1.2
Tuberculous oophoritis	4	0.8
Granulosa cell tumor	4	0.8
Theca cell tumor	3	0.6
Dysgerminoma	3	0.6
Mixed theca and granulosa cell tumor	2	0.4
Brenner cell tumor	2	0.4
Teratoma	2	0.4
Seminoma	1	0.2
Ovarian pregnancy	1	0.2
Plexiform angioma	1	0.2
	Total 472	

Figure 1

important in another and its removal may make the patient's final condition worse than the first.

From the standpoint of a surgical pathologist the ovary is the most surgically mistreated organ of the body. It is a mystery to me why surgeons should regard with such indifference the castration of the human female. If the male testicle were attacked with the same impunity our courts would be overflowing with law suits. Cystic ovaries have too long been considered a cause of such vague complaints as abdominal pain, menstrual disorders, neuropsychiatric states, with removal justified for these diagnoses. This is indeed a fallacious assumption. Pathologists should refrain from diagnosing such things as multiple follicle cysts, corpus luteum cysts, retention cysts, perioophoritis, ovarian hematomas, fibrotic ovaries, and interstitial ovarian fibrosis because they appease the conscience of the operator but do not justify the surgery. Most surgeons have a blind faith in the infallibility of pathology and when they see these terms under the diagnosis they accept these as pathologic and cut.

There is one difficulty and that is that the microscope does not reveal function and therefore all problems must be settled by coordinated study between surgeons, endocrinologists, and pathologists. Pain is one of the most common causes or excuses for attacking the ovary. Aside from lesions which irritate or stretch the peritoneum I do not know of a single ovarian lesion constantly accompanied by pain which is permanently relieved by removal of the ovary. True, some of the physiologic processes of the ovary are regularly attended with pain such as the midcycle pain from the rupturing follicle but who would advocate oophorectomy for this? Cranial operations for the relief of simple headache would be just as sensible. Many surgeons have had the experience of seeing a polycystic ovary at operation and after leaving it alone the pain ceases. The disturbance was obviously physiologic and the polycystic state of the ovary had nothing to do with the pain clinically observed. Ovaries showing every stage of physiologic anatomic development that have been erroneously interpreted as pathologic have been unmolested at surgery and the pain has ceased. Every pathologist in the course of routine autopsies

	Before education 1939-1945		*After education 1946*	
Private Hospital No. 1				
Total number ovaries removed	622		147	
Total with pathologic lesion	175	28.1%	97	66%
Total with no pathologic lesion	447	71.8%	50	34%
Private Hospitals No. 2, 3, 4 and 5				
Total number ovaries removed	485		162	
Total with pathologic lesion	143	29.4%	82	51%
Total with no pathologic lesion	342	70.5%	80	49%
County Hospital				
Total number ovaries removed	74		16	
Total with pathologic lesion	40	54.1%	14	90%
Total with no pathologic lesion	34	45.9%	2	10%

Figure 2

discovers all of these physiologic stages which have never caused the patient a single symptom.

In the performance of its predestined function the ovary is periodically forming cysts and one which does not show cysts is either aplastic or senile. Each maturing follicle forms a "cyst," and when ruptured a hemorrhagic "cyst" or "tumor" results, which in turn is followed by a "fibroma" or a "corpora albicans" with the end result of an "interstitial fibrosis" or "interstitial oophoritis."

There are essentially only two types of physiologic changes in the ovary, the cystic and the fibrotic. The cystic results either from the distention of a maturing follicle or from the rupture of this follicle with hemorrhage into the cyst. In most instances this is followed by a corpus luteum and finally fibrosis. Nature's way of terminating the menstrual cycle is by a generalized fibrosis. These deviations in anatomic structure are without association of a real pathologic process. When a surgeon sees these fruits of his labor displayed in a pathologic museum he continues to attempt to relieve his patients of something that never caused them trouble in the first place.

It is my opinion that an ovary should never be removed unless it is truly pathologic or unless it is removed along with other organs in the attempt to cure a carcinoma. It is generally true that the younger a woman is the more severe the menopausal symptoms will be following castration. Nevertheless some of the most severe reactions are observed in women who had ceased menstruation.

This discussion plus a demonstration of the physiologic ovarian changes was presented at the staff meetings of the various hospitals concerned.

As can be seen from the statistics in Figure 2 the desired effect was forthcoming. Today the great bulk of ovaries removed in the hospitals I serve as surgical pathologist actually have a pathologic lesion. I am sure the same end result can be accomplished in any hospital merely by presentation of a similar demonstration prepared locally.

SCIENTIFIC EXHIBIT

Chronic Shock: The Problem of Reduced Blood Volume in the Chronically Ill Patient

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IT HAS long been recognized that the debilitated patient is a "poor risk" for extensive surgical procedures. More specifically, such patients have little or no tolerance for blood loss, heal their wounds slowly and suffer a generally protracted convalescence. Recent studies have shown that a reduced blood volume is a significant feature of the nutritional depletion of the chronically ill patient. Quantitative restoration of the blood volume by transfusions of whole blood effectively prepares the patient for an aggressive surgical program. A graphic summary of these observations is presented in the following charts and diagrams.

Definition: Chronic shock is the syndrome of weight loss, reduced blood volume and increased interstitial fluid volume observed in the chronically ill patient.

Surgical Significance: Reduced tolerance for surgical blood loss, impaired wound healing, and protracted convalescence.

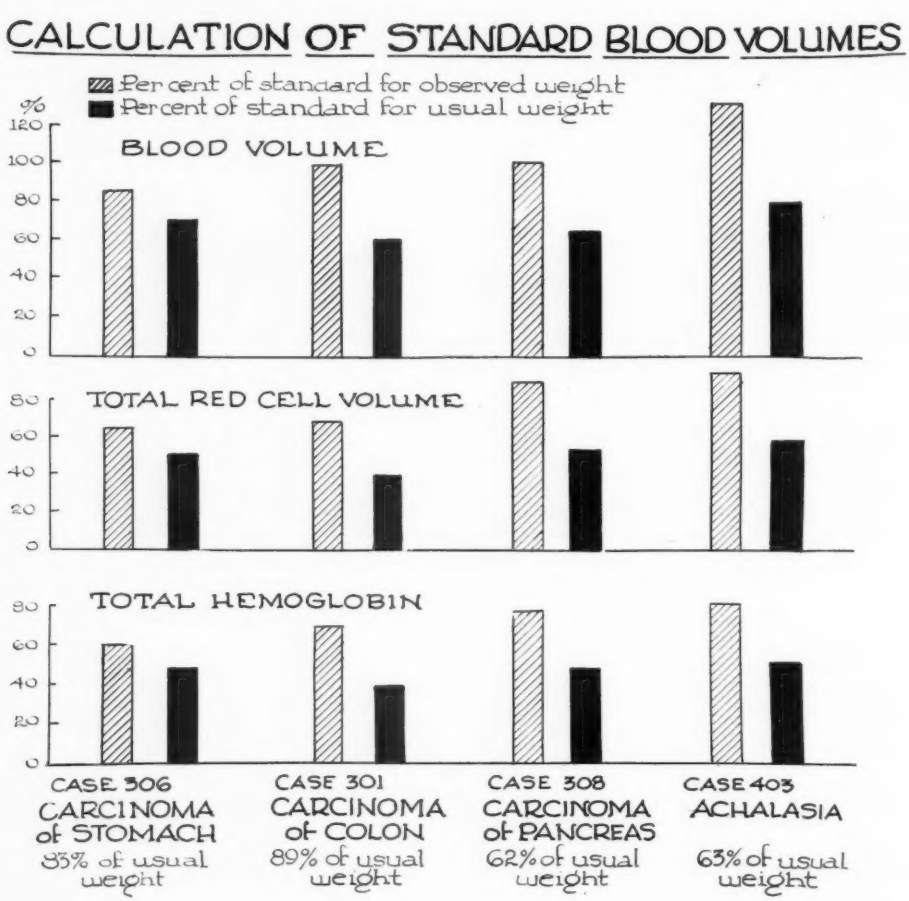
Adapted from an exhibit awarded "Honorable Mention" by the American Medical Association.

This is publication No. 7 from a series of studies being conducted under a grant from the Research and Development Division, Office of the Surgeon General, United States Army.

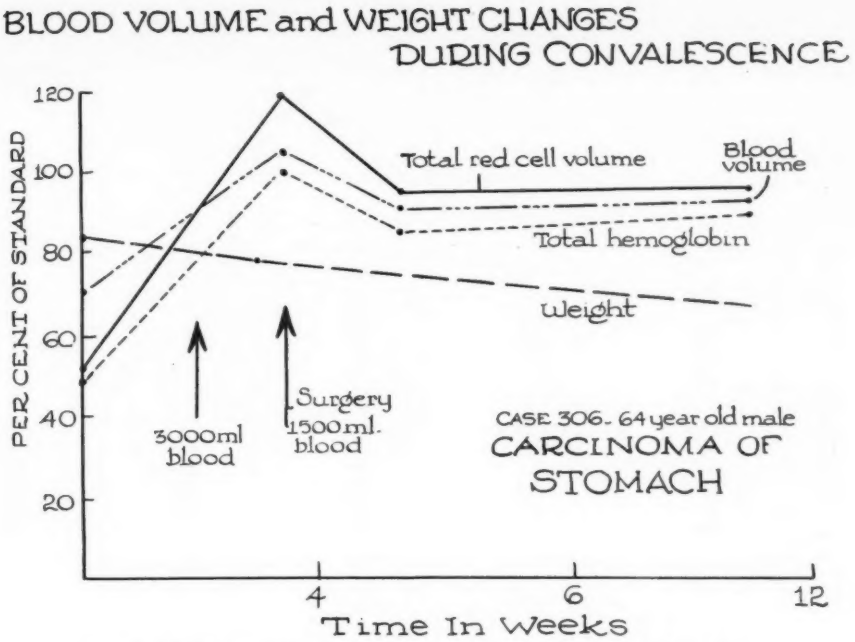
Method of study used in compiling data on 100 debilitated patients. Total blood volume has been determined by the plasma-dye-hematocrit method, using the blue dye T-1824. Total red cell volume, total circulating hemoglobin, and plasma protein were calculated from blood volume.

Calculations of deficits are made from following standard values:

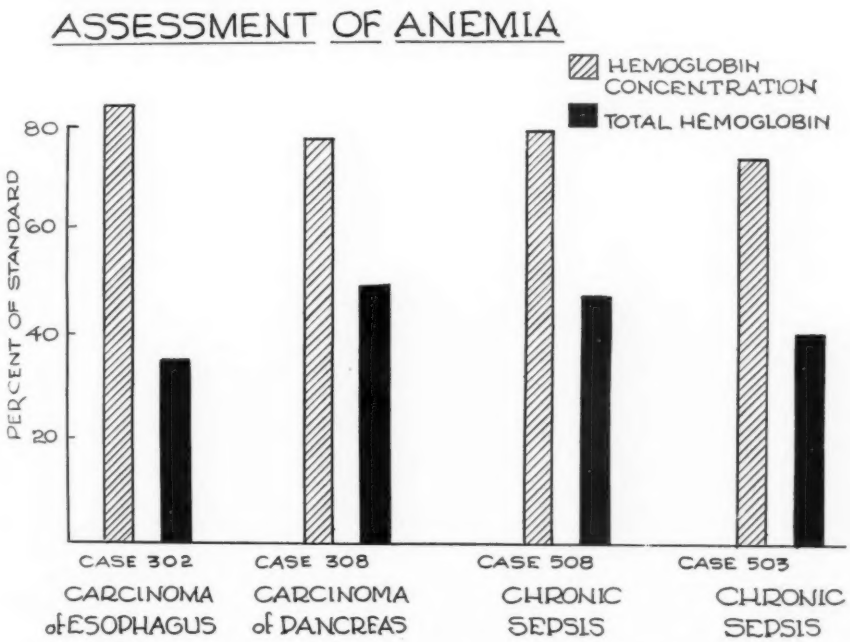
Total blood volume	85 ml. per kg.
Plasma volume	45 ml. per kg.
Cell volume	40 ml. per kg.
Total circulating hemoglobin	12.75 gm. per kg.



The usual weight in health is used in calculating standard blood volumes and deficits. The blood volumes of patients with weight loss may be approximate standard values based on observed weights, but are distinctly below standard values based on usual weights in health.

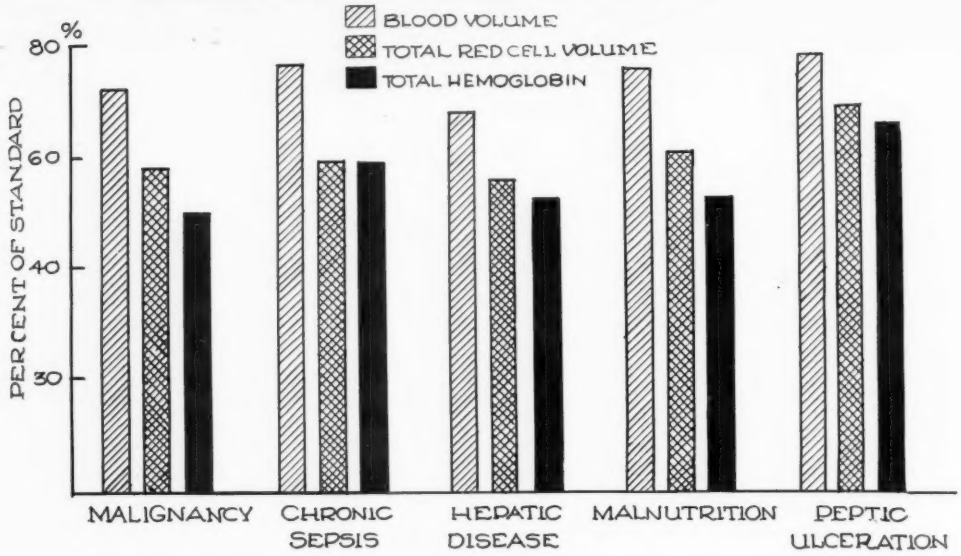


Clinical finding supporting the calculation of standard blood volumes on basis of usual weight in health: (1) This level is maintained during convalescence after transfusion and appropriate surgical treatment. (2) Restoration of blood volume to standard values is accomplished without significant hemoconcentration.



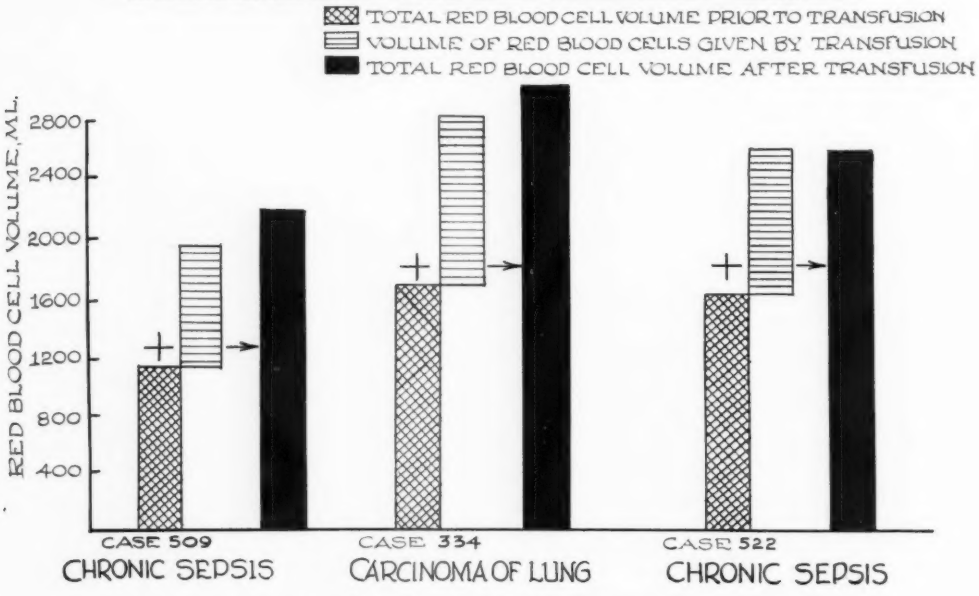
Hemoglobin concentrations, red cell counts, and hematocrit percentages fail to reflect the true deficiency of total red blood cell mass apparent after blood volume determination.

FINDINGS IN CHRONIC SHOCK



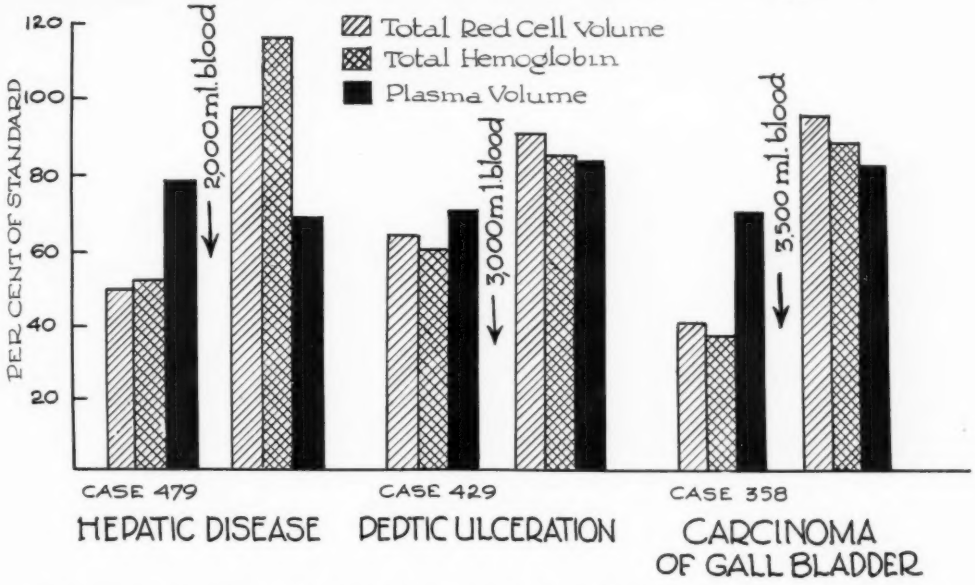
In chronic shock: (1) The total blood volume is low. (2) The total circulating red cell volume is low. (3) The total amount of circulating hemoglobin is low. There is no correlation between amount of weight lost and observed deficits.

DIRECT APPROACH TO THE PROBLEM — WHOLE BLOOD BY TRANSFUSION



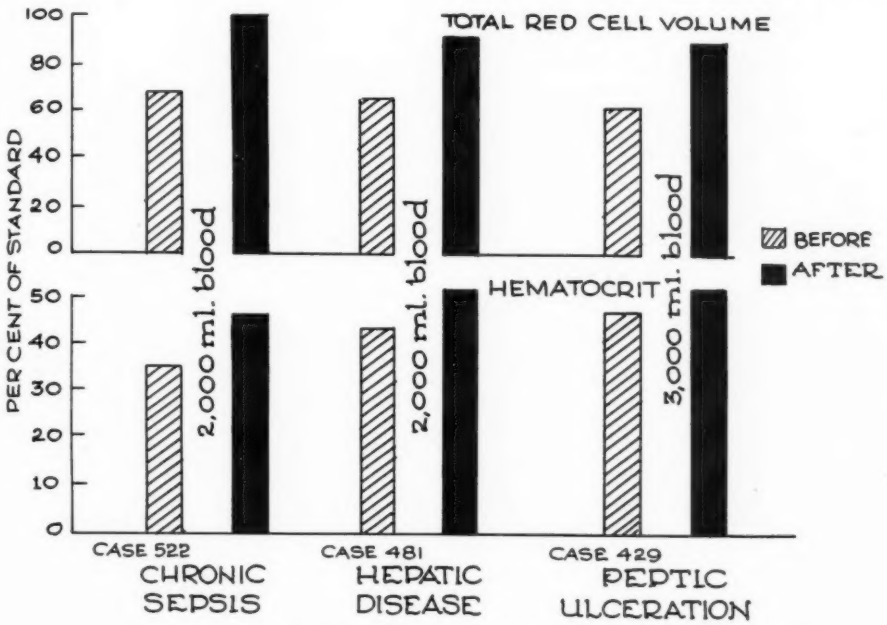
In absence of continued bleeding, transfusion replacement of red blood cells is quantitatively effective.

RESPONSE TO TRANSFUSION THERAPY



The average quantity of blood required to restore the total red blood cell mass was 2,700 ml.

RESTORATION WITH NO SIGNIFICANT HEMOCONCENTRATION



After restoration of total red cell volume to standard, the hematocrit seldom exceeds 50 per cent. Restoration of plasma volume occurs later during convalescence.

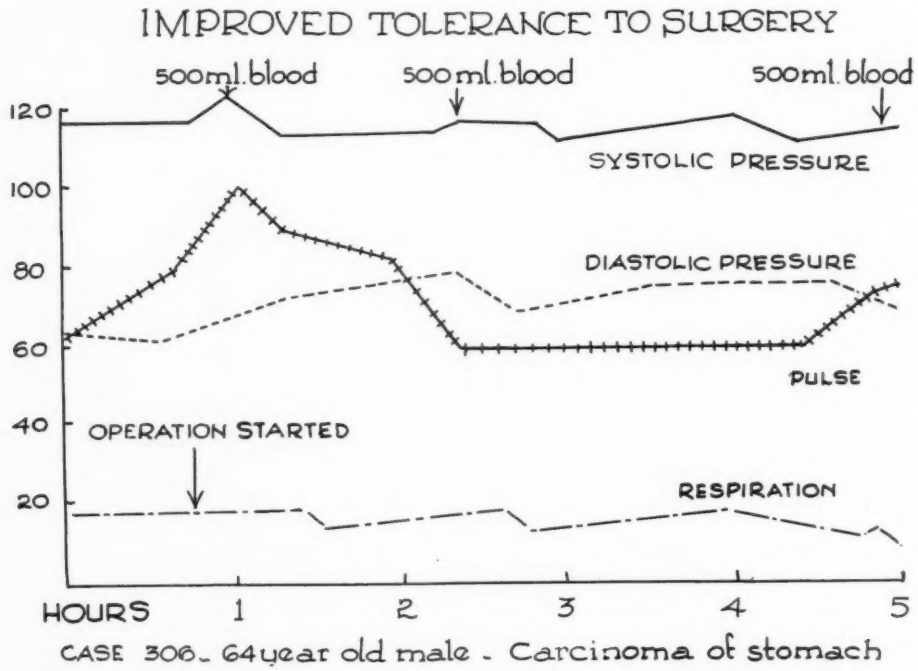
ESOPHAGEAL STRICTURE

Following esophagogastrectomy for malignancy (March, 1946), gastrostomy, July 5, 1946
Rapid clinical recovery following dilation

Case 326—57-year-old male; usual weight, 220 lb.

Date	*Period*	*Weight (pounds)*	*Total Hemoglobin*	*Total Cell Volume*	*Blood Volume*
			Percentage of Standard		
July 1, 1946	Preoperative; prior to transfusion	149	56	65	73
July 5, 1946	Preoperative; after transfusion of 2,500 ml. blood	Unchanged	113	118	126
July 17, 1946	Postoperative; high protein gastrostomy feeding	Unchanged	90	90	78
August 27, 1946	Postoperative; patient able to swallow high protein diet	161	100	102	98

After effective transfusion therapy, restoration of depleted reservoirs of tissue protein is accomplished by feeding a complete diet.



Restoration of total red cell volume to standard has prepared patients for all indicated surgical operations. Whole blood transfusions provide adequate and rapid preoperative preparation and insure a smooth operative course. Blood lost during operations is replaced concomitantly.

Anesthesia: *Cyclopropane and ether by endotracheal technic.*
Operation: *Total gastrectomy and anterior gastroenterostomy for carcinoma with subtotal pancreatectomy, splenectomy, transverse colectomy, resection of greater omentum, and jejunostomy.*

The Medical Bookman

PRACTICE OF ALLERGY*

WARREN T. VAUGHAN'S classic text on the Practice of Allergy has been published in a new edition revised by J. Harvey Black of Dallas, Texas. This second edition was in preparation at the time of Dr. Vaughan's untimely death in 1944. As the readers of the original edition will recall, the book had a flavor unusual in medical volumes. It made fascinating reading for physicians in all specialties and the second edition seems to have lost none of the interest and flavor of the earlier work. Dr. Black has been wise in retaining most of Vaughan's phrasing and attack. The text has been altered, however, whenever new developments in the field required it.

The volume is notable in several respects. A thirty-six page bibliography includes most of the articles of real importance in the field. The controversial subjects of bacterial allergy and focal infection are masterfully handled, the controversies adequately presented, the various points of view in the literature well covered, and the author's own views with reasons therefor clearly stated.

The section describing the part which fungi play in allergy covers sixty pages and is beautifully illustrated by photograph and sketch. In addition to the discussion of respiratory allergy to these agents, a chapter on fungus infection of the skin with associated allergy has been rewritten by Dr. J. B. Howell of Dallas, Texas, and represents the present thinking of the dermatologist and the allergist.

Allergy to insects and their products, called in this text Entomogenous Allergy, is briefly but interestingly covered, the various reports in the literature dealing with sensitization to bed bugs, mosquitoes, fleas, house fly, butterfly, caddis fly, and other insects being reviewed.

Most of the drugs capable of acting as allergens are listed in the section on drug allergy. A short section descriptive of the manifestations usually produced by each drug is included. One wishes that more space were given to the treatment of drug allergy by desensitization in those instances in which this measure is required. The subject is covered in the text by the following statement, "The same general principle is followed as in all other desensitization processes, namely, gradual increases from an original minute dose." On the whole, however, the text throughout is most thorough and complete, each section including discussion of all related factors.

Despite this completeness the reviewer notes certain omissions. Relatively little mention is made of the relationship of allergic symptoms to the physiology of the autonomic nervous system. It is apparent in the study of any allergic patient that there is a very close interplay between allergic symptoms and the patient's emotional state. Several psychiatrists have commented at length upon the characteristic personality background or traits of persons suffering from allergic manifestations.

Since the work of Dale and Lewis of England, it has become increasingly apparent that histamine or some closely related substances such as acetylcholine are the substances liberated at the site of allergic reaction. The liberation at these sites then produces the phenomena characteristic of these drugs, that is, capillary permeability, capillary dilatation, and edema. For some years it has also been recognized from the work of Dale and others that acetylcholine is the substance which when liberated at the end organs of parasympathetic nerves, produces the parasympathetic response. It appears then that the same or similar chemical substances can be liberated in tissue by two means. It would seem reasonable then that adding histamine-like substances from a parasympathetic nerve response to the histamine already formed at a given site from the allergic reaction will push the histamine reactivity above the threshold necessary for the production of physiologic alterations in that tissue and thereby give rise to the allergic manifestations.

The text also fails to bring out a point which the reviewer holds essential: that the diagnosis of the allergic state be precisely made before the difficult, time-consuming job of detecting allergens is instituted. Searching for allergens in a non-allergic patient is no more reasonable than starting deep

*Practice of Allergy. By Warren T. Vaughan, M.D., and J. Harvey Black, M.D. 2nd Edition, 1132 pages, illustrated. 1948, C. V. Mosby Company, St. Louis. Price $15.00.

CHOCOLATE. COCOA.—*Theobroma cacao*

Chocolate is a contribution from the New World, Mexico, the West Indies, and elsewhere. It is now grown also in British West Africa, Brazil, Ecuador, San Domingo and Venezuela. Cortez first knew of cocoa when he was treated to the drink from gold cups in the palace of the Aztec Emperor, Montezuma. The Spaniards carried cocoa back to Spain. They kept its secret for many years, selling it at a high price, as chocolate to the wealthy classes in Europe.

The word cocoa is a corruption of the Spanish cacao which is in turn an adaptation of the Indian Cacauatl. The Mexican Indian also used the term chocolatl. As early as 1550 chocolate factories were scattered through southern Europe. The first factory in America was established in 1765.

The cocoa tree grows from 16 to 30 feet in height. The pods are large, from 6 to 12 inches long and from 2 to 5 inches in diameter. The cocoa bean, about the size and shape of a kidney bean, is protected by many layers, from inward outward, the bean shell, slime tissue, a soft inner shell and the hard almost woody outer shell of the pod.

The roasting of the bean adds flavor. It is put through several processes before it emerges as a thick oily liquid which is run into molds and allowed to cool. This is the common bitter chocolate. In sweet chocolate cocoa butter and sugar are added. Milk chocolate contains, in addition, a condensed milk or milk powder. As stated under soy bean it may contain soy bean milk and oil.

Cocoa, *Theobroma cacao* or breakfast cocoa, is derived from the same bean but has more of the cocoa butter or oil removed.

Cocoa butter is used in confectionery, especially in chocolate covered candies, in the drug trade and in the manufacture of toilet preparations and cosmetics. It is especially appropriate for some of these uses since it melts at about body temperature. It does not turn rancid.

A boy with dermatosis, allergic to chocolate, who had never had asthma, developed bronchopneumonia. During convalescence he ate chocolate. His cough persisted for three or four weeks. The blood showed 12 per cent eosinophilia.

Chocolate was then eliminated from the diet after which the cough promptly disappeared and the eosinophilia dropped to 4 per cent. I have seen cases of dermatitis from cocoa butter applied locally, in persons who were atopic to chocolate.

Example of manner in which description of foods is covered.

x-ray therapy for carcinoma before the diagnosis of carcinoma has been established. The text further allows a misconception common to American physicians to be preserved. This misconception is that the diagnosis of the allergic state is made by applying skin tests. It is a surprisingly common and a completely false belief that by the application of skin tests one may learn whether or not the symptoms of which the patient complains are allergic in origin. One finds frequently that people who have never in their lives had allergic manifestations will show several positive skin tests, and it is conversely not infrequent that patients suffering from frankly allergic symptoms will give completely negative skin reactions. The skin test is a tool in the armamentarium of the physician, giving him clues as to which substances may be weighted more heavily and which substances may be regarded more lightly in patients in whom the diagnosis of the allergic state has been positively made by other means. Despite the failure of deemphasis of skin testing, the text does discuss the failure of the skin test method and the various possible reasons therefore.

Another omission apparent to the reviewer is the relatively brief discussion of sensitization to oils and fat. This omission is probably due to the lack of knowledge of and to the dearth of literature on this subject. It is frequently and sometimes embarrassingly apparent, however, to those studying this field that tissues do become sensitized to substances of this class. One would have enjoyed having this relatively dark corner of the subject of allergy illuminated. However, to the reviewer's knowledge the subject has not been studied sufficiently to allow fuller discussion.

Another subject which is but briefly mentioned is cardiac asthma. Among patients with cardiac decompensation it is not infrequent to see patients whose dyspnea is due obviously to narrowing of the bronchial airways rather than to pulmonary edema or to diminished oxygen and increased carbon dioxide tension. Among these people one occasionally finds those patients in whom specific agents effect allergic responses, the exhibition of these increasing or their removal decreasing such symptoms. More frequently, however, one is totally unable to find any allergenic factors operating. It is suspected that some reflex mechanism precipitated by the physiology of cardiac failure may be operative in these patients producing bronchial edema, hypersecretion of bronchial mucous glands, or constriction of the bronchial musculature. However, one searches the literature for adequate discussion of this subject without avail.

Turning to consideration of the various sections,

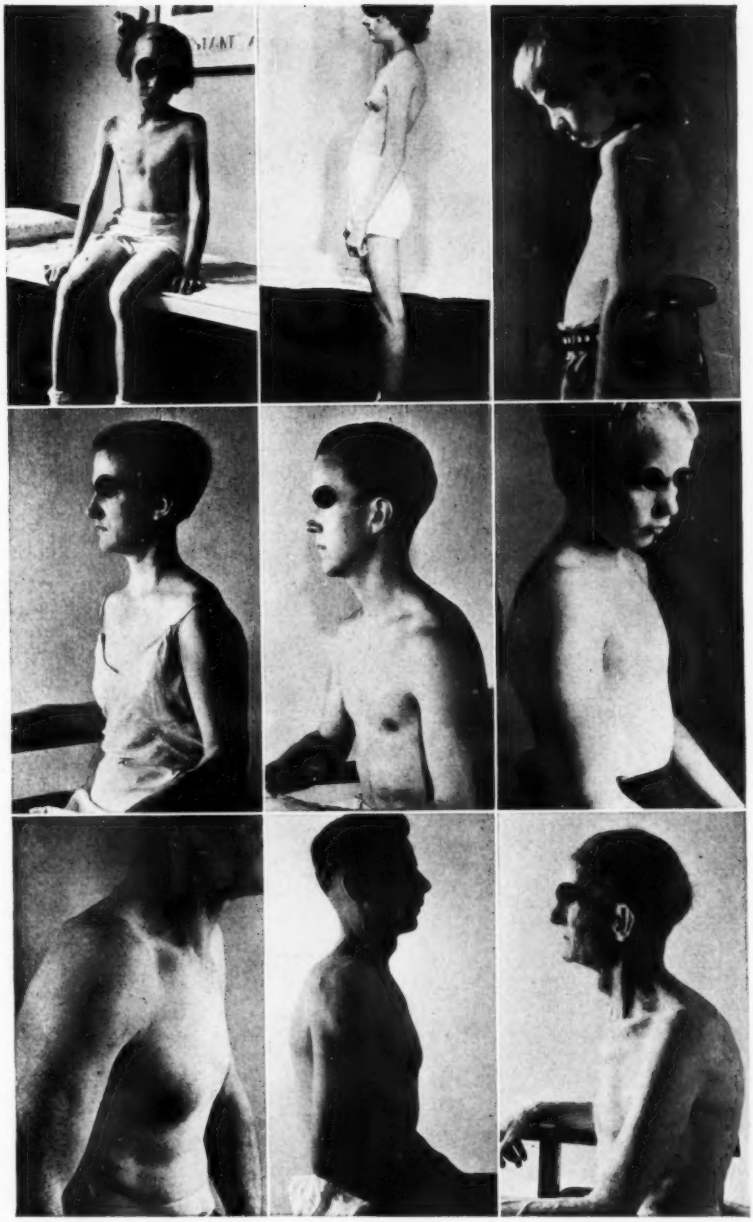


Figure 304. Types of chest deformity associated with asthma.

First row: Flat chest with tendency to pigeon breast, seen especially frequently in the growing child. Generalized muscular asthenia is commonly present. Improvement in muscle tone, with graded exercises if necessary, often helps the general condition.

Second row: With beginning barrel chest, the clavicle becomes raised, appearing horizontal rather than dipping downward toward the sternum. Owing to the chest deformity and the allergic facies, depression of malar prominences, the first two figures of this row (not related) resemble each other to such an extent that they might be taken for brother and sister.

Third row: More pronounced emphysema with barrel chest and horizontal clavicles.

one finds that in discussion of the basic sciences as related to allergy, the authors have made romantic a study which for many is strictly pedantic. All of the concepts and theories which have been held by other observers are entertainingly presented. The authors discuss their own theories making plain that they are theories and voicing the opinion that as more adequate concepts are derived out of bet-

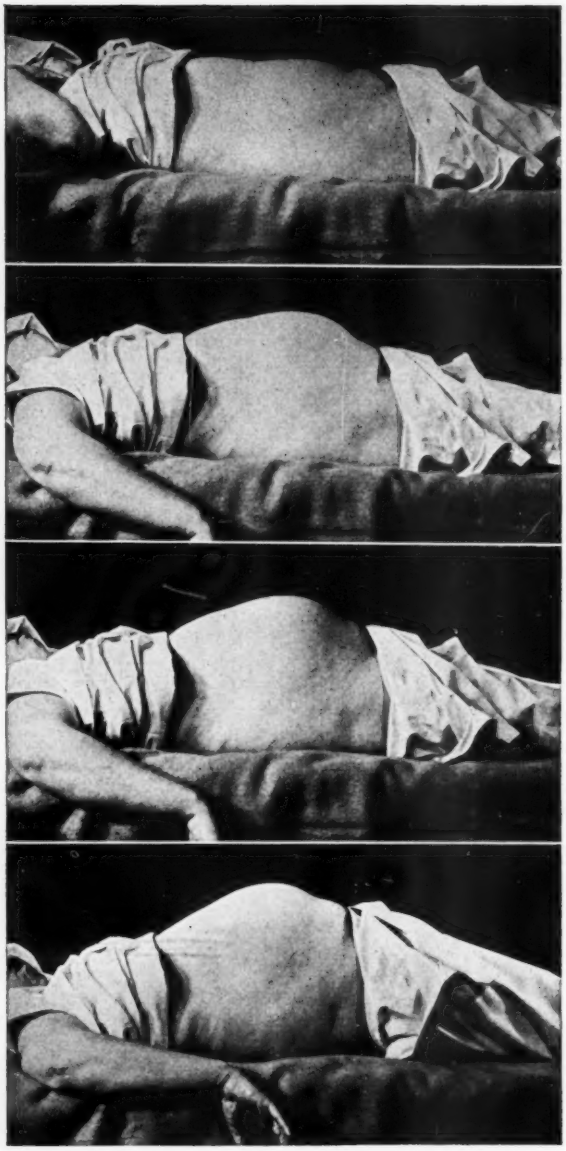


Figure 333. The nervous or psychic factor in allergic responses. This patient's abdomen would balloon up, as is shown here, within three minutes after eating certain foods, especially orange juice, or after dipping the hand in ice water. Photograph was taken with the latter as the excitant. Breath holding or other obvious diaphragmatic manipulation did not accompany this response. The back is not arched. The ballooning persisted although the patient continued to breathe normally. Relief followed dipping the hand in hot water.

Abdominal distention is a frequent feature of gastrointestinal allergy. However in the above case the extreme degree and rapidity of response suggests a predominant psychic factor. The patient was not of the hysterical type. Allergic food avoidances have given relief through the two intervening years. Allergic therapy relieved, possibly through suggestion.

Illustrations from: Vaughan-Black, "Principles and Practice of Allergy."

ter experimental evidence these theories will then be replaced.

The sources of information regarding a patient's sensitization are thoroughly covered. Many helpful points are made in regard to history taking. It is brought out that the important points of the allergic's history can be arrived at only by intimate and painstaking discussion with the patient in re-

gard to how he lives, what he lives with, what his food habits are, what are the idiosyncrasies of all kinds which he has experienced. All of the methods of obtaining information from skin testing are carefully evaluated, including the method of passive transfer and the method of making mucous membrane tests. Patch testing is discussed together with the substances to which the method is applicable.

The chapter on the leukopenic index remains. This method was devised by Dr. Vaughan but is not at present widely used. It depends upon the depression of the total neutrophile count over one or two hours in a patient who has ingested a specific food immediately preceding the test. No discussion is made of the eosinophilic index, a method similar to the leukopenic index but depending upon the elevation of the total eosinophile count following such ingestion. Neither is mention made of Arthur Coca's contention that ingestion of an allergenic food will cause tachycardia. Coca feeds the patient a dish of a given food recording the pulse rate at frequent intervals during the course of the next two hours. If the pulse rate rises during this time Coca assumes that this represents evidence of sensitization. This method has never been accepted by American allergists but neither has the concept been refuted. The book includes a useful portion on methods of testing for physical allergy including cold testing, heat testing, actinic testing, and trauma testing with a review of some of the observations made by various observers in regard to this subject.

The volume's essential core is the sections dealing with the diagnosis and treatment of food allergy and with the study of pollens and pollinosis. These chapters comprise about half the text and are the high points therein. No other English text covers these subjects comparably in either breadth or depth except perhaps Coca, Walzer and Thommen in their monograph published in 1931. All of the methods of detecting allergenic foods are adequately and interestingly discussed. The section on botanical description of the food plants, their origin, their occurrence and use in manufactured or prepared foods is exhaustive and the best available. Likewise the pollen section is just as thorough. Part of it is written by and the rest contains much material from the student who has done the greatest work in this field, O. C. Durham. One actually recognizes the grasses and plants from the excellent photographs made by the late Dr. Vaughan in person.

One concludes that, though flaws are apparent, this volume remains a classic in its field and has grown in statute with revision. It is unequivocally recommended alike for the casually interested and to the student of the subject. Each will find reward in plenty.

A. W.

New Books Received

► GENERAL ENDOCRINOLOGY - - -

By Donnell Turner, Ph.D., Associate Professor of Zoology, Northwestern University. 1st Edition. 604 pages with 164 figures. 1948, W. B. Saunders Company, Philadelphia and London. Price $6.75.

Even more than the title implies, this text discusses the subject of endocrinology from a broad didactic point of view. The author reveals an extraordinary ability to integrate the fundamental knowledge of this subject from the various biologic sciences and to transmit this information in an orderly and clear manner. The subject matter is approached from an experimental rather than from a clinical point of view. As the text is intended for students who are concentrating on experimental biology, the material is presented as a fundamental aspect of biologic science rather than as a medical specialty. However, the author has chosen the human being wherever possible to illustrate the operation of biologic principles.

Aside from the unquestionable importance of this book to students of experimental biology, the book should be of value to medical students and graduate physicians who are interested in obtaining the broadest point of view in the study of endocrinology, and for those practicing physicians who desire a refreshing review of the basic knowledge in this field.

M. S.

► CLINICAL UROLOGY - - -

By L. E. McCrea, M.D. 2nd Edition, 503 pages, 263 illustrations. 1948, F. A. Davis and Company, Philadelphia. Price $6.50.

According to the preface, this book is to aid in the determination of "what is it? what to do? and how to do it" in the form of a concise, practical treatise. It is intended for general practitioners, interns, and students, and is said to have been largely rewritten.

On the whole, it achieves its objectives, since it covers the field pretty completely and concisely, and is unusually well illustrated (the author is an enthusiastic amateur medical photographer). However, a book intended for the group mentioned above should certainly contain a few key references to the literature where there are grounds for differences of opinion. Unfortunately, this one does not. Better proof reading would have eliminated certain amusing inconsistencies, such as "impassable strictures are treated by internal urethrotomy . . ." (you just *can't* do it!), and "the infected prostate can and does not act as a focus of infection," to name just a couple.

Your reviewer also objects to devoting almost two pages to a classification of "Neurogenic Bladder," a poor but widely used term, while giving only a few lines to a fairly ambiguous discussion of treatment. One must also criticize his recommendation of mercuric cyanide as an antiseptic (it ain't) for sterilizing catheters, and of a combination of prostigmine and Syntropan, which ought to counteract one another pretty completely, for postoperative retention of the urine.

Much of the book is much too dogmatic to be of maximum value, although this doubtless results from the desire to be brief. Thus "a stricture should never be cut if it is possible to pass a filiform through it," and "exposure of the tumor (in the bladder) should always be by the suprapubic route" need a good deal of qualification and discussion.

Even so, the book could be recommended to "students, interns, and general practitioners" were it not for the author's puffing up of the long discarded old superstition that sudden emptying of the chronically distended bladder is dangerous, and his recommendation that anuria from sulfonamides be treated by three to four thousand cubic centimeters of isotonic saline by vein each day. This is not good modern treatment because its use in an anuric patient is so likely to lead to edema and to cardiac failure. C. D. C.

► PSYCHOSOMATIC DIAGNOSIS AND TREATMENT - - -

By Flanders Dunbar, M.D., with the assistance of Drs. Jacob Arlow, Raymond Hussey, Bertram Lewin, Robert C. Lowe, Sydney Rubin, E. Schneider, Lester W. Sontag, and members of the staff of the Department of Medicine and Psychiatry of Columbia-Presbyterian Medical Center, New York. 501 pages. 1948, C. V. Mosby Company, St. Louis. Price $6.50.

Proceeding on the theory that there are psychophysiologic interrelationships, not only in the visceral diseases, but also in the nervous and infectious diseases as well, the authors have applied certain "psychodynamic principles previously studied in visceral diseases to syndromes involving voluntary musculature and reactions to reactions." They conclude that each type of disease syndrome is probably characterized by a specific type of personality profile and that, therefore, it is necessary to "study the whole organism," including history-taking as "a vital and integral part of the study of the personality profile."

► FRACTURES AND DISLOCATIONS - - -

By Edwin O. Geckeler, M.D., Philadelphia. 4th Edition, 371 pages. 1948, The Williams & Wilkins Company, Baltimore. Price $5.00.

This edition, which was written for the general practitioner and the student, brings up to date the new developments in traumatic surgery since the third edition was written, including methods which developed out of experience gained in the recent war. Where there is a choice of procedure in the management of various fractures, the author continues his earlier practice of recommending the method he considers the simplest and most reliable. Each section of the book is profusely illustrated.

Men of Medicine

THE CLASS WILL COME TO ORDER

DR. ERNEST SACHS, holder of this country's first professorship in neurologic surgery, is a plumpish, balding, straight-talking man whose ability to make the air in an operating room cyanotic is vividly recalled by former students from England to Argentine.

The great saving grace about this pioneer brain surgeon is that virtually all of his colleagues and many of his students realize his peppery, impatient, and outspoken qualities have been prompted through the years by a fierce devotion to basic principles.

In a specialty which requires profound concentration, Dr. Sachs has long been known for the demands he has made of himself and those around him in the provision of meticulous care for the patient. Always methodical, he has insisted that it is the duty of the surgeon himself to prepare his own operating field. Throughout the three decades since his professorship at Washington University in St. Louis was established, he has stoutly maintained that not alone the neurologist, but the neurologic surgeon must participate in a diagnosis if intelligent treatment is to be rendered.

Even those former students, who once in a while disrupted Sachs' celebrated weekly, Thursday noon diagnostic clinics by setting off alarm clocks, freely concede now that his stern attention to business produced deep-seated and lasting benefits. Of those clinics at the St. Louis medical school, Dr. Sachs is wont to say, in the words of Dr. William Osler, one of his own great teachers: "They were to train the powers of observation. Their purpose was to train the eye that sees not, the hand that feels not and the ear that hears not."

That his lessons, delivered in a strong voice which carries with the force of a foghorn, were effective is evidenced in the achievements of some of the students and fellows who worked under him. Among these are Drs. Alton Ochsner, Cobb Pilcher, Edward Fincher, J. Barrett Brown, Roland M. Klemme and Henry G. Schwartz. The latter succeeded Dr. Sachs when he retired as chief of neurologic surgery at Washington University in July, 1946. Of the Sachs-trained fellows who have remained in surgery, not one has failed to be named chief of the specialty at his institution, including Oxford and the Universities of Liverpool and Argentina. Seven hold professorships.

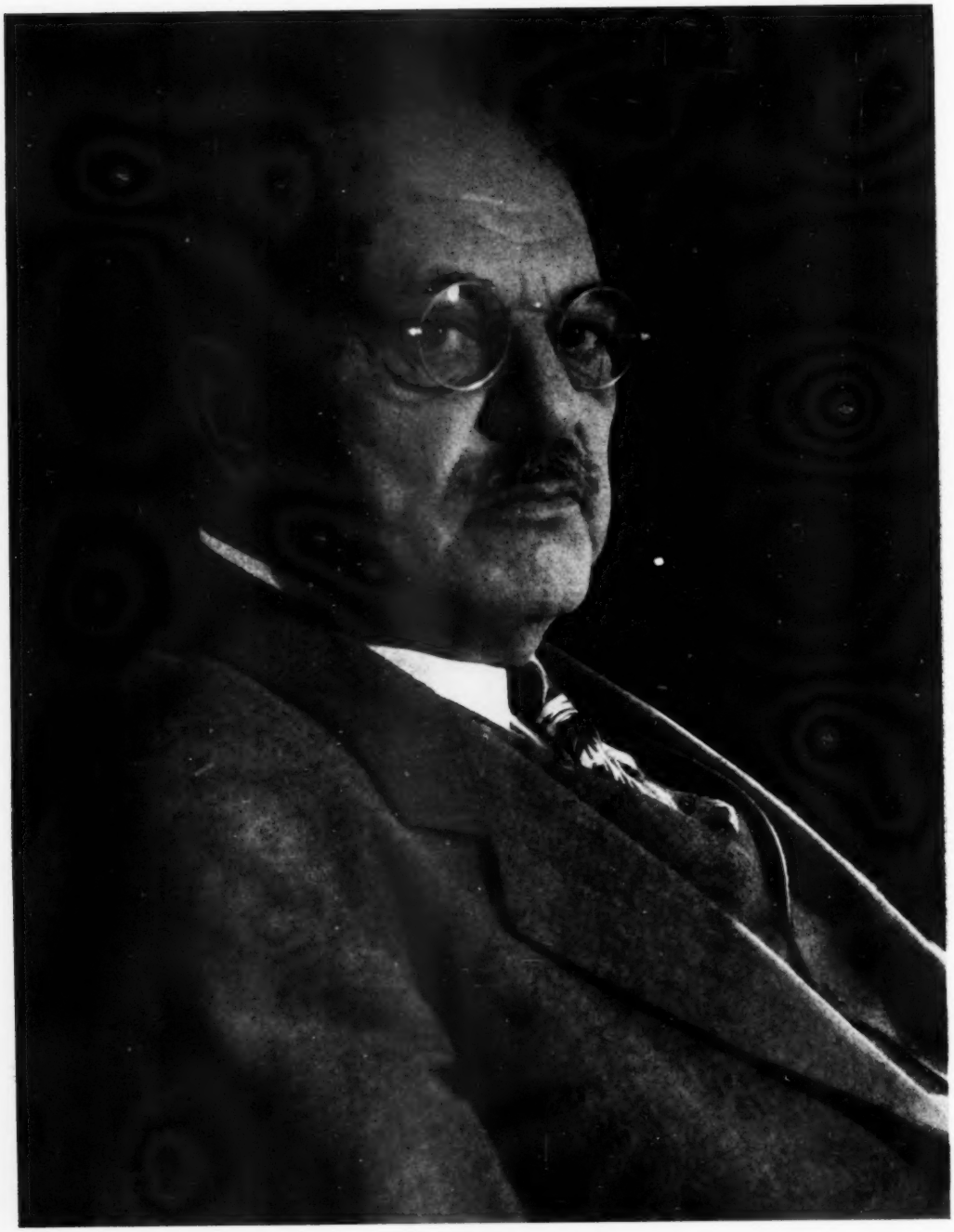
Above all, Dr. Sachs counts himself a teacher. Seventy years old last January, his temper has become quiescent and his manner has mellowed. Yet he still can whip up a disturbance at the thought that, to his knowledge, not a single U.S. medical school includes the fundamentals of pedagogy in medicine in the material it gives its students.

Himself a product of a family noted for its achievements in education, he believes as he looks back upon his career that he strove always to avoid the old, stereotyped questions, to lead students to do their own thinking. Too often, he long has maintained, this principle has been cold-shouldered by instructors appointed only for their accomplishments in the laboratory and the operating room, without regard to their ability as teachers.

"You can't teach only by Hippocrates and the grace of God," he is prone to say. "The outstanding student doesn't need deft instruction, but the majority of students are not outstanding, they are average. A great deal can be done, I believe, to improve the character of teaching in our medical schools. Improve that and we will improve the quality of service rendered by the average practitioner."

As Ernest Sachs looks about at the prevailing scene in the field of neurologic surgery, he confesses that the sight leaves him somewhat incredulous. When he began, in the first two decades of the century, it was a lonely realm in which Harvey Cushing, Charles H. Frazier, Charles Elsberg and he were about the only figures.

Not until 1919, shortly before Sachs, Cushing



ERNEST SACHS

and others formed the Society of Neurological Surgeons, was the specialty fully created. "I often wonder how we got by," Dr. Sachs says in awed tones. "Even blood transfusions were troublesome. It was a rare sight to see a patient with a brain tumor. Before 1918, and Walter Dandy's discovery and development of ventriculography, our clinics thought they were doing splendidly if they localized 50 per cent of tumors. Finding young men willing to enter the field then was vastly more difficult than finding an adequate supply of nurses is now."

Today, he notes, hundreds of gifted, young students are streaming into neurologic surgery. Something is considered amiss if existing tumors are not successfully located in 97 per cent of the craniotomies performed.

Dr. Sachs spent his childhood and early manhood in his native New York. For a while, he confesses, he had a trying time because he attended the private school of which his father, the late Dr. Julius (Ph.D., not M.D.) Sachs was principal. "Have you ever been the principal's son?" the physician inquires ruefully, his direct gaze coming over his spectacles. The senior Sachs later was to be, for fifteen years, professor of secondary education at the Columbia University Teachers' College. There is now, at the college a scholarship endowment fund established by the surgeon's father and his mother, Rosa Sachs.

THE family lived on New York's West side, near the Museum of Natural History, but Dr. Sachs stoutly maintains its proximity had nothing to do with his early interest in science, biology and medicine. Always a plump youngster who insists he never was called "Fatty," he was ever the serious, studious type. His boyhood collections of rocks and butterflies were the bane of his mother's existence, but young Ernest, with the dogged application that was to be a permanent hallmark of his character, made weekly excursions into the countryside to add to his store of specimens.

When he was 7, he began taking cello lessons. "It was the fashion," he recalls a trifle wryly. Unlike most juvenile musicians, however, Ernest stuck to his cello. Later, at Harvard (where he was class of 1900), he was to organize a Haydn String Quartet; one of his fondest college memories is the occasion when the quartet gave its first concert at the dean's home, creating such interest that Charles Eliot Norton was moved to attend.

Propelled by an early interest in surgery, he visited the Massachusetts General Hospital one day during his senior year, to see whether he could tolerate watching an operation. It was an especially gory amputation of the knee joint he viewed on that occasion, and he was compelled fully six times to dash out for air. Happily for neurosurgery, he never suffered this malaise after he got to medical school at Johns Hopkins in the autumn of 1900.

With characteristic enthusiasm, Dr. Sachs counts himself lucky to have had the teachers he did. It was the period of Osler, William S. Halsted and Arpad G. Gerster. To the latter, especially, Dr. Sachs credits his abounding love for the principles of postoperative care, and the notion that a man may be an excellent operator, yet still be woefully deficient in his knowledge of the many niceties of patient care.

When Dr. Sachs entered his period of internship at Mount Sinai Hospital in New York, his objective was general surgery. But his uncle, Dr. Bernard Sachs, who headed the neurology department at the hospital, began impressing on the young intern's consciousness the fact that there was an acute shortage of personnel in the virgin field of neurologic surgery. Everywhere the word was, "Brain tumors always die." In the profession, or more exactly, in a tiny segment of it, there was alert interest in the work of Harvey Cushing, just then beginning to apply to brain and spinal cord surgery the principles promulgated by Dr. Halsted for general surgery—delicate care of the tissues, control of hemorrhage, the use of the silk technic.

Sachs gave in. "I don't know," he says reflectively, "I suppose I went in for neurosurgery because it was a field everybody said was so 'hopeless.'" He performed his first brain operation at a New York nursing home. "It was a tough case," he recalls candidly, "and I don't think I was so damn calm."

Even then, he was convinced that sound neurosurgery demanded, first of all, a thoroughgoing knowledge of neurology. Without this, he reasoned, there never could be a neurosurgical specialty. He therefore spent about eight months studying neurology under Henry Oppenheim and Heinrich Obersteiner in Berlin and Vienna, and followed this with two years of study under England's Sir Victor Horsley. These three, and especially the latter, Sachs counts among his idols, but it is no uncritical idolatry. In later years Dr. Sachs was to

Photograph taken at the annual meeting of the Society of Neurological Surgeons, New York, about 1936. Seated, from left to right: Charles A. Elsberg, Charles H. Frazier, Howard Naffziger, Harvey Cushing, and Alfred S. Taylor. Standing, left to right: Byron Stookey, Wilder Penfield, Loyal Davis, Samuel Harvey, Kenneth G. McKenzie, Max M. Peet, Charles Bagley, H. H. Kerr, Carl W. Rand, Ernest Sachs, J. Jay Keegan, Jason Mixter, Charles E. Dowman, Francis C. Grant, and W. McK. Craig.

note disapprovingly that Oppenheim's day sanctioned separate, preliminary diagnosis by the medical man and then operation by the surgeon.

Results in those days were so discouraging, and candidates for the field so few in number that Dr. Sachs has recalled that his first neurologic contact with Dr. Cushing in 1909 was in the form of a letter received while he was with Sir Victor. Dr. Halsted had just given permission for a resident, Dr. Cushing wrote wistfully, and would Dr. Sachs like to come home and take the job? Sachs regretfully had to turn down the offer because his work with Dr. Horsley was not yet finished.

A few years later, Dr. Sachs himself was to receive a note from Dr. Frazier suggesting that the former take on a promising young comer named M. M. Peet. "He was too promising," Dr. Sachs now exclaims. "He never came." As late as 1919, he was to hear again on the same subject from Dr. Cushing, who wrote to ask if Dr. Sachs knew of any worthwhile potential assistants. In sum, even the neurosurgical topnotchers were scraping around at the bottom of the barrel.

When Dr. Sachs returned to New York from England in 1909, he hoped to enjoy a period of general surgical practice, but pressure in another direction was too strong. Very shortly thereafter, he was (as he puts it) "pushed" into neurosurgery; after stints in Montefiore, Beth Israel and Bellevue Hospitals, he joined the Washington University medical school faculty in 1911.

There, an old student remembers, he quickly established a custom he never forsook in the ensuing years, of accompanying students and fellows to brain cases in personal rounds he made at least once a week. He thought it a major offense then, to smoke in a patient's room, and he still does.

During those first years in St. Louis, a suspiciously high number of patients were reported by their physicians to be dying of "a stroke." The pudgy Sachs frame and full moustache began popping up at meetings of county medical societies in

Missouri and Illinois, as the surgeon started to beat the bushes in an effort to spread awareness of the symptoms of brain tumor. Dr. Sachs was a little tired of having everybody remember the Maine. He wanted more practitioners to remember the tumor.

ACTUALLY, the history of neurosurgery began in 1919. It was the year in which Dr. Will Mayo first introduced the term, "neurologic surgery," after Dr. Cushing had delivered a memorable address on brain tumor statistics before the American College of Surgeons. "Today, we have witnessed the birth of a new specialty," was Dr. Mayo's comment. It was the year, also, in which Dr. Sachs received his epoch-making professorship, now a commonplace in most universities.

Dr. Sachs, ridden by a deplorable weakness for not remembering faces but blessed with a remarkable memory for facts and a methodical way with old correspondence, has rich recollections of those first days of the Society of Neurological Surgeons.

At the initial meeting, Dr. Cushing was to operate on a brain tumor, Dr. Sachs recalls, but when the patient was wheeled in, he was spewing, coughing, and blue. After a period of watching, while the anesthetist, who had given ether, vainly tried to quiet the patient, Dr. Cushing suddenly said, "We won't operate. Any patient who behaves as he does won't stand operation."

The decision, a hard one to make, exerted a profound impression upon Dr. Sachs, who has stubbornly emphasized the precept to more than a generation of students that the patient comes first. Only one other violation of his code makes him come out of his corner with greater belligerency.

That is to find a surgeon, especially a neurologic surgeon, who gives up on a case, even the most desperate. The only progress recorded to date, he feels, has been achieved because somewhere along the line there were men willing to keep trying no matter how high the odds.

Dr. Cushing was the first president of the Society of Neurological Surgeons and Dr. Sachs its first secretary, a post he held for almost six years. Later, he too, became president, a post he also was to hold in 1943 with the American Neurological Association. He also is an honorary member of the Royal (British) Academy.

In an address delivered three years ago to the Surgeons, Dr. Sachs recalled that its first years were filled with much technical correspondence and difference of opinion on matters which today provoke only unanimity. Dr. Sachs' early correspondence with his friend Cushing dealt with Gigli saws, different kinds of operative clips, a type of electric light bulb which Dr. Cushing had seen Dr. Sachs use on a headlight and could not get in Boston. Comments at the 1921 meeting were critical of ventrical punctures and the withdrawing of cerebrospinal fluid to reduce pressure. Dr. Cushing was worried about the possibility of serious accidents resulting from Dandy's procedure for injecting air into the brain to locate tumors.

Although Dr. Sachs did his own first air injection in 1918, shortly after Dr. Dandy had described the technic, he confesses it was almost a decade before he really got off the fence. Today, he regards this and the introduction of electrosurgical technic by Dr. Cushing in 1927 as the major signs of neurosurgery's coming of age.

Friends say the fact is that, progressive teacher though he may have been, Dr. Sachs tempered his enthusiasm with conservatism for the benefit of the patient. He has a stubborn passion for things that are tried and true. Though it remains the bane of his operating room assistants, he still sterilizes his hands with potassium permangenate and oxalic acid before operating.

Never one to shirk from objective self-analysis, Dr. Sachs candidly describes himself and many of the other pioneers of neurosurgery as "a bunch of prima donnas." In 1923, he and a number of the others temporarily fell into disrepute with the hard-working Dr. Cushing when they arrived at the society's annual dinner, mellowed by a cocktail session, only to have Dr. Cushing announce, soon after the meal: "Time to go, men. I've arranged a clinic for you at 8 o'clock, and the patients are all lined up."

"Nobody wanted to go," Dr. Sachs recollects with a sigh. "Looking particularly at the secretary—me—Dr. Cushing said angrily, 'Well, if you're going to make this an eating club, I'm going to get out.'" Next day, however, Cushing was affability himself, staged an impressive operative demonstration and even had a friendly word for the disgraced secretary.

Dr. Sachs, as he likes to put it himself, has been "guilty of" two books of considerable stature. The first, "Diagnosis and Treatment of Brain Tumors," was published in 1931 and became the first English language work detailing this field in comprehen-

sive fashion. He wrote it primarily with a view to helping the general practitioner recognize the presence of brain tumors and grow ever more aware of the old Sachs rallying cry, that brain tumors were not rare diseases.

His second book, "The Care of the Neurosurgical Patient," published only four years ago, summed up what Dr. Sachs has stood for: meticulous attention to detail.

Dedication of his first book, and a graceful tribute in the preface of the second one to his wife, Mary Koues Sachs, are more than a formal gesture from husband to spouse. In point of fact, some friends say, only those students lucky enough to get one of the invitations to the Sachs home and to see the surgeon in his home habitat got a good look at the real measure of the man, under the iron curtain of his pedagogic crispness.

They still call each other "Sweetheart," after thirty-six years of marriage.

Their close kinship serves to dispel any notion that Dr. Sachs may have a constitutional objection to the marriage institution when he says regretfully that distractions from duty inevitably ensue for the high proportion of today's interns who are married. "When I was young," he is wont to say sternly, "a man couldn't get a hospital place if he was married."

Mary sachs is her husband's junior by four years, a slight, slender, white-haired, effervescent woman who writes prize-winning poetry and has been active in the League of Women Voters and other politically-conscious organizations. Friends call her "Maisie." She knows her own mind and, some say, has been unsuccessful until recent years in only one effort involving her husband; this was her steady campaign to make him professionally mellow. From the beginning of their union, she has been her husband's literary editor and critic. "She's always felt I needed one," Dr. Sachs sighs resignedly. "And she's so right."

Save only for patient's names, she has shared Dr. Sachs' complex cases with him. With startling ease, she can explain the significance of his contribution in 1946, when he helped pioneer the handling of brain abscesses by excision as a means of reducing mortality from draining. She is familiar with the details of his important 1932 report on effective uses of electrosurgery, especially in the treatment of blood vessel tumors in the brain.

"Mary is an incredible optimist," Dr. Sachs says ruefully. "She had to be, to live with me." He takes a curious kind of pride in the fact that they were married only four months after they had met, in the Adirondack Mountain town of Keene Valley, N.Y., where the Sachs family had a summer home and where Mrs. Sachs then was working as a settlement house social worker.

Today, at the stage of his career where he is getting professional longevity plaudits from the *Journal of Neurosurgery*—such a printed posy was sent his way in the January issue—Dr. Sachs nevertheless continues to adhere to his old schedule: breakfast at 7 a.m., first operation at 8. More often than not, he continues to be the first to reach the operating room where his "Do It Now!" signs became a fixture many years ago. Save for his retirement from teaching, his only concession to the passing years has been a diminution in the number of operations he performs; until recently, he observed a six-day operating week. As usual, Mrs. Sachs never permits her husband to get off, of a morning, without first fixing his breakfast.

Sharper than ever is the contrast between Sachs, the stern operating room disciplinarian and Sachs, the healer orienting a patient, or a patient's family. He continues to have an active fear of becoming one of those physicians who barricade themselves behind secretaries, hence startles many a telephoning patient by picking up the receiver himself, as soon as it rings. He has an unrequited penchant for tinkering. Recently, when he moved his office in mid-town St. Louis, he did most of the picture-hanging himself and also made a special point of reserving for himself the dubious privilege of installing a pair of glass window-draftbreakers which he carried into the office one day.

If unhurried, his office memoranda turn up written in a fine, legible hand, but if they have been turned out under pressure, his handwriting is well-nigh undecipherable, not only to his staff but frequently to himself. Talk of an interesting case still engrosses him to the point of dramatization; when he dictates an operative note, he relives it, complete with gestures. He is as careful in his dress and habits as he is in his surgery. "There can be a tornado coming in over the horizon, but that will make no difference to Ernie Sachs," a colleague observes smartly. "He will take time to answer all of that day's letters and straighten out the top of his desk before leaving for cover."

Balancing some of the ex-students who vow that

Dr. Sachs was an operating room knuckle-rapper are those whose conscientiousness and aptitude earned them a startling amount of help, when it came time to obtain fellowships or professional entrée.

Even more, there is the downright reverence in which Dr. Sachs is held by his secretary and his anesthetist. The former, Miss Gertrude Moyle, has been with Dr. Sachs for thirty-two years. She never tires of recalling how he took an entire morning personally to observe an operation on her aged mother, and how from time to time he has driven to her home in St. Louis' south side to deliver a few tulips. Most of the 3,000 operations Dr. Sachs now has performed have had the assistance of his anesthetist, Miss Jessie Lindsey. To both of them, Dr. Sachs is "The Chief." Miss Lindsey remains in awe before his operating room dexterity, although he often has noted emphatically, "It's more important to be careful than swift."

Of the 3,000 operations, incidentally, about 1,500 have been for the removal of tumors. They helped Dr. Sachs create for the Washington University medical school one of the world's largest pathologic collections.

Miss Lindsey contributed a revealing tribute to Dr. Sachs in a letter written for a volume Mrs. Sachs compiled among colleagues and students to present to her husband on the occasion of his 70th birthday.

"If you never have worked for the Chief, you don't know what a privilege it is to work for someone who inspires you to do your very best," the anesthetist wrote. "If others care more for the disease and its scientific aspects than for the patient, let them! But don't you! Just learn to care more particularly for the individual patient than for some special feature of the disease, for the Chief never forgets the patient in his interest in the tumor itself.

"Even in a hopeless case, where there is little to do except keep the patient comfortable, he does not throw up his hands and turn it over to the house staff or the interns. No sir, not even the dressings. Patients always get the best from him—sometimes food from his own house or perhaps a doll or a toy especially longed for. Nothing is too much trouble.

"If you want to see the Chief at his best, watch him as he handles some poor child with a recurrent tumor—a hopeless case. He does not give up. Even though he cannot give the family one thread of hope to hold on to, and even after he has told them the truth without any sugarcoating, he himself will stand up and defy the Grim Reaper, doing everything in his power to save his patient.

"Another neurosurgeon asked the Chief, 'Do you operate on glioblastomas?' When the Chief answered, 'Yes, we do,' the other asked, 'Why?' Dr. Sachs looked straight at the doctor and said, 'Everyone has a right to live as long as he can, and be as comfortable as possible while he is living. And, too, you are never sure what you are going to find until you look in.'

"Now that I have put down a bit of the Chief's philosophy as he lives it, all I say is that I am glad I work for him," Miss Lindsey concluded earnestly. "The world needs more men like him."

Dr. and Mrs. Sachs live in the three-story, eight-room brick house in suburban Clayton, Mo., which they have occupied since 1914. They rattle around in it a bit, now that their two sons have gone. The older boy, 32-year-old Dr. Ernest Sachs, Jr., has enhanced his father's happiness by deciding to become a neurosurgeon and currently is studying at the Lahey Clinic. He served in the army as an assistant surgeon in the European Theatre. Son Thomas Dudley, named after one of Mrs. Sachs' ancestors who was the first governor of Massachusetts, is a 23-year-old senior at the University of California. A navy veteran, he also plans to study medicine, giving the family a 1000 batting average.

In his younger days, Dr. Sachs was a mountain-climber, taking Mrs. Sachs away from her poesy to scramble up some peak in the Adirondacks or the Alps. By now, of course, that avocation regretfully has gone by the boards, as has the string quartet which Dr. Sachs organized and kept active in St. Louis much as he had at Harvard. Acquisition of a hothouse has helped the surgeon pursue his fetish for wearing a flower in his buttonhole whenever possible.

Dr. Sachs vows that his enthusiastic efforts in gardening and orchid cultivation have proved none too successful thus far. "I'm a dub," he exclaims. He usually manages to add hastily, "But you should have seen how darn well I did with tomatoes last summer at Keene Valley. Turned out a gallon of tomato soy, believe it or not."

Still built along plump lines, he constantly is resolving to watch his diet, but never does. He

dotes on fish dishes, likes Sherry and Rhine wine, can whip up a very tasty apple popover. In his youth, he was an inveterate pipe-smoker, but for many years his pipe-smoking has been reserved for summer walks in the hills. "Gad, I'm almost a saint!" he cries in sudden realization, as he explains that he gave up ordinary smoking to accommodate Mrs. Sachs' allergy.

Both the surgeon and his wife are extremely fond of opera and theater and of reading. A mite apologetically, no doubt because the jacket drawings shock him, Dr. Sachs confesses that he usually spices his medical reading with biographies and book-club selections. His personal library numbers 5,000 volumes.

It has been a long, long road to Tipperary for neurosurgery since Dr. R. J. Godlee performed the first brain tumor removal in November, 1884. Dr. Sachs terms "phenomenal" the advances which have been wrought during his lifetime, and in which he has played so active a part.

"What is the future of neurologic surgery?" he asked in the conclusion to his 1945 book. "Can it be further developed and perfected, or have we about reached the end of possible achievement?"

Providing young men in the field do not permit themselves to become mere technicians, Dr. Sachs could come up with but one answer.

"The possibilities for improvement are almost limitless," he wrote. "By means of technical advances alone, tumors and other types of lesions will be dealt with successfully, and I feel sure that certain brain tumors which today (1945) are treated inadequately, will be removed successfully or, if not removed, will be destroyed by other means.

"The recent work of Dandy on the treatment of intercranial aneurysms is an example of one new field. The pioneer work of Peet, Adson and Smithwick in the treatment of hypertension by sympathectomy is a further advance in surgery of the sympathetic nervous system, and this is but an opening wedge for the study of other uses for this type of surgery.

"The surgical treatment of myasthenia gravis by the removal of a thymoma is still another virgin field which, when understood, may throw new light on the function of the thymus gland. Even some of the degenerative diseases of the nervous system, such as multiple sclerosis and the muscular dystrophies, may respond to surgical measures."

He added, prophetically, "We certainly have little conception as yet of the possibilities that may be opened up when radioactivity is combined with the powerful action of the cyclotron.

"The indisputable proof that neurologic surgery is continuing to broaden its scope lies in the fact that increasing numbers of young men are even now entering this ever widening field."

One day recently, after Dr. Sachs had been regaling his wife with the details of an intriguing new case, she turned to him suddenly and exclaimed, "Sweetheart, thank the Lord I'm not married to a starch and blueing salesman!"

To this, even after forty-five years of exacting practice, Ernie Sachs said a prompt and heartfelt Amen.

Robert H. Schulman

EDITORIALS

TASK FORCE MEDICINE

FAR-REACHING in its significance and of the deepest concern and absorbing interest to those in the medical profession is the recent task force report on Federal medical services, published by the Hoover Commission on Organization of the Executive Branch of the Government.

The 16-member task force committee, which included 11 members chosen because they are medical authorities, did not investigate compulsory health insurance, ruling that this issue was beyond the scope of its inquiry.

What the committee did do was to make an exhaustive survey of existing Federal medical facilities and to recommend sweeping reorganization of these services.

The report was amply documented with facts to support its basic thesis, that the principal need to correct existing faults is an overall plan. The committee pointed out that, first, a clear definition of the extent of the government's responsibilities in the medical field must be drawn by Congress, and, second, an organization to carry out the foregoing commitment must be drafted. The committee's fundamental recommendation is that Federal subsidies be directed to building up medical schools and community and private hospitals, with a minimum of government supervision.

The report makes a sharp distinction between military and non-military medicine, with the recommendation that general hospitals of the armed services in which dependents are served, and all medical and hospital installations of the Veterans Administration, be integrated into the civilian health program.

In releasing the report, Herbert Hoover, chairman of the commission which directed this and other task force studies, made it clear that task force findings in any field do not necessarily represent the final conclusions of the commission.

Under existing conditions, the committee said, the government is assuming uncalculated obligations without an understanding of their ultimate cost, the lack of professional manpower to execute them, or the adverse effect on the hospital system of the nation.

Substantiating this judgment, the committee reported that in the New York area four Army and Air Force hospitals could be closed, with a resulting reduction in medical officer requirements of 80 to 85 per cent, and the further advantage of providing a better standard of service by such a move.

Recommendations of the report are based on the assumption that a Cabinet post of health, education, and welfare will be created to take over the functions of the Federal Security Agency. The committee excluded from its consideration the possibility of a separate Cabinet department for health alone. The report specified, however, that if such a health department were instituted, the recommended organization could be adapted to such a plan.

As suggested by the report, a national bureau of health would be established, headed by a doctor of medicine who would report only to the Cabinet secretary, with the proviso that no administrative under-secretary should be a physician.

Within the bureau there would be three divisions: (1) medical care, (2) public health, and (3) research and training. The medical care division would assume all Federal hospitalization and direct care, including the general hospitals of the military establishments and all medical facilities of the Veterans Administration.

Present functions of the Public Health Service would be pared down in the proposed public health agency to retain only administering grants-in-aid in that field, and in addition, the new division would then take over the Food and Drug Administration and run an administration clearing house.

The projected research and training agency would take over the research program of the National Institutes of Health and the clinical research center project, activities which have been administered by the present Public Health Service.

Work of the Federal Children's Bureau, the report recommends, should continue as a unit under the secretary of the proposed department, except that within three years its specific programs on maternal aid and crippled children should be transferred to the new bureau of health.

The present method of the Veterans Administration in securing part-time services of private specialists was enthusiastically approved by the committee, with the recommendation that this system be expanded as far as possible under the proposed set-up. An end to the commissioning of officers for the Public Health Service was recommended as a step to facilitate the carrying out of the above personnel method. It was recommended that all military medical procurement be done by one branch, under a medical officer.

Congress must make certain fundamental policy decisions in regard to the national health system, the report pointed out. Number one is the question of whether medical service should be given to all veterans, or, in the case of non-service connected disabilities, only to those in financial need. A voluntary health insurance plan for veterans was suggested, calling for government payment of premiums where the veteran cannot pay. Also put up to Congress were the questions of whether civilian dependents of military personnel are entitled to free medical care and how long disabled officers should be cared for in military hospitals.

J. S.

REHABILITATION IN THE MANAGEMENT OF CARDIOVASCULAR PROBLEMS

THE PERSON who suffers from a disabling cardiovascular disease must be considered as much a candidate for rehabilitation training as the one who has been stricken by any other type of physical disability. Though a somewhat different approach may have to be followed in handling cardiac patients, optimum results can be obtained if evaluation and conditioning training are begun as soon as feasible after the acute phase of the illness has subsided, and are done under strict medical surveillance.

According to Dr. Howard A. Rusk, studies from many sources have shown that patients observed after prolonged bed rest show a decreased blood volume, a decrease in the size of the heart, an inability to do sustained work, and a loss in reaction time. There was also a marked loss of vitamins in the urine. It was also found practically impossible to keep patients in normal calcium balance the first six weeks of bed rest regardless of diet or medication. It also required more than twice as much protein in the diet to keep the patient in positive nitrogen balance.

It has been noted clinically that when patients requiring bed rest are given conditioning exercises for the unaffected parts as soon as it is medically feasible, these deconditioning phenomena are markedly decreased, and, in some instances, obviated. When the Army Air Forces Rheumatic Fever Control program was organized during the war, 85 per cent of the patients with rheumatic fever were of necessity discharged from the Army as unfit for further military service. A program of planned convalescence was initiated, starting with bed exercises under close supervision a week after the temperature subsided, and gradually increasing until the end of the fifth month when the patient had a full thirty days of eight-hours-a-day on-the-job training on the air base at which he was hospitalized.

Dr. Rusk advocates the need for basic re-

search in criteria for evaluating the capacity of individual patients to work. As an example, he has called attention to the energy requirements in using the bed pan in the ordinary hospital bed, as compared with the energy expended in taking one or two steps to a commode, and has pointed out that in actual practice most of the leading cardiologists have discarded the bed pan routine in almost all instances, although, by custom, it is still practiced in many hospitals during the early rest period in coronary thrombosis. Before arbitrarily discontinuing this practice, there is need for basic study of such energy requirements in order to put this practice on a factual basis.

With proper management, many chronic cardiac patients progress favorably on a reasonably active regimen. The physician must, of course, be explicit in his instruction and suggestions to the patient, so that whatever precautionary measures are required will be observed.

Rather than assume that the person who has a chronic cardiac disease must perforce lead an inactive, dependent existence, rehabilitation studies at Bellevue Hospital in New York City have shown that very often such "disabled" persons, when placed in jobs commensurate with their reduced physical capacity, establish excellent employment records, showing even greater production rates, and lower accident and absentee rates than employees who are not in any way incapacitated.

To estimate the individual's capacity for work, it is necessary to observe him at work, and to determine how he goes to and from work. It is often necessary to advise a change of occupation and mode of transportation—and sometimes of residence—so that there will be no undue taxing of the patient's strength and energy. For most patients, it has been found that work—of the proper sort and suited to the individual—is one of our most valuable therapeutic tools in the management of all types of chronic disease. A. E.

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To provide the general practitioner, as well as the specialist, with concise, readily available information on the latest, proved methods of diagnosis and treatment of conditions commonly encountered in an average practice, POSTGRADUATE MEDICINE offers this department to its readers. It is not, of course, intended to present these discussions as the only acceptable therapeutic procedures to be used, but rather to offer simple regimens and recommendations based on the extensive experience of the physicians who prepared these summaries.

HEMORRHOIDS - - -

HEMORRHOIDS or "piles" are tumors made up of normal and abnormal elements of radicals of the superior, middle, and inferior hemorrhoidal veins and perivascular tissue. They are found beneath the mucous membrane of the lower rectal segment, anal, and perianal skin.

CLASSIFICATION

Internal, external or combined internal-external. The majority are combined. External skin tabs are not true hemorrhoids.

ETIOLOGY AND PATHOLOGY

Hemorrhoids are essentially varicose veins and are associated with some degree of infection. The portal of entry of such infection is through the anal crypts. The inflammatory process resulting in the production of hemorrhoids is a perivascular process differing from the intimal process found in the lower extremities in the production of varicose veins.

Increased venous pressure in the veins of this area resulting from the upright position of the body, the absence of valves in the veins, and the constricting influence of the bowel musculature itself accentuated by pregnancy, constipation, chronic catharsis, increased intra-abdominal pressure from any cause, obstruction of the portal system, heart disease, pelvic and rectal tumors may be factors in the production of hemorrhoids.

Following the introduction of infection, the diseased vein walls rupture, giving rise to all stages of inflammation, thrombi, fibrosis, edema, or gangrene.

SYMPTOMS

Varying degrees of bleeding, pain and protrusion.

Clinical

1. External Hemorrhoids

a. Simple—Appear as engorged veins beneath the anal margin. Straining or squatting may be necessary for demonstration.

b. Thrombosed—Vary from single, small, thrombosed pile to massive thrombosis and edema. Edema of anal margin may predominate.

c. External skin tabs—Fibrous folds of skin at the anal orifice. Result most commonly following hemorrhoidectomy or from a thrombosed external hemorrhoid.

d. Differential diagnosis—External hemorrhoids should be differentiated from nonsyphilitic condyloma, varying types of ulcers, such as tuberculosis and epithelioma.

2. Internal Hemorrhoids

a. Simple—Found proximal to the pectinate line and visualized by anoscope or proctoscope; may be overlooked on palpation because of soft, rubbery character.

b. Complicated—Prolapsing, thrombotic, or ulcerated.

c. Differential diagnosis—Internal hemorrhoids should be differentiated from hypertrophied papillae and rectal polyps.

TREATMENT

General Measures—The presence of hemorrhoids does not necessarily warrant treatment. Hemorrhoids causing symptoms should be treated unless there are obvious contraindications. Conservative measures are usually indicated for the acute phases.

1. External Hemorrhoids

a. Simple—Uncomplicated external hemorrhoids are ordinarily associated with internal hemorrhoids and should be treated by excision as combined hemorrhoids.

b. Thrombosed external hemorrhoids—Small, discrete clots not causing symptoms may subside without surgical excision. Pain, however, is usually associated and may be extreme. Excision of clots with overlying skin is desirable. This may be done as an office procedure following injection of the thrombosed external hemorrhoid with 1 per cent procaine.

Massive, thrombosed, external hemorrhoids with varying degrees of edema are best treated conservatively until the edema and infection have subsided. Surgical excision is usually indicated later. Bed rest, elevation of the buttocks, wet dressings with applications of extremes of hot or cold are desirable. Ointments, salves, and suppositories are of little value.

NONOPERATIVE TREATMENT

1. Internal Hemorrhoids

a. Simple, uncomplicated internal hemorrhoids may

frequently be treated satisfactorily by injections.

Patients who complain of bleeding without pain or excessive protrusion, who, on examination, present internal hemorrhoids and who do not exhibit evidence of other disease as demonstrated by stricture or pain on examination, can usually be given relief from their symptoms by the injection method of treatment.

Two solutions have been used most commonly for the injection treatment of hemorrhoids. A 5 per cent quinine and urea hydrochloride solution is probably the most popular and safest. One or two hemorrhoids at a time can be injected with 1 to 2 cc. of the solution in each pile.

The needle is thrust into the substance of the internal pile above the pectinate line and the solution injected, symmetrically distending the pile. This produces a perivascular inflammatory process, resulting in shrinkage of the hemorrhoidal mass as healing and fibrosis take place. Usually, two to three injections at intervals are given into each hemorrhoidal area.

Phenol, 5 per cent, in a vegetable oil solution, such as almond oil, can also be used. Smaller amounts of the solution are injected into each pile. Longer intervals should elapse between injections but the resulting fibrosis in the hands of some has been reported as more satisfactory than that resulting from quinine and urea.

Attention to bowel regulation and other hygienic measures for the general welfare of the patient will materially improve results of this type of treatment.

b. Complicated internal hemorrhoids. Internal piles associated with thrombi, infection or with any other type of anorectal disease should be removed surgically.

c. Complications of the injection method of treatment. Superficial sloughs may occur from excessive pressure on the overlying mucosa. Pelvic phlebitis, abscess formation with fistula and other complications are rare but they do occur. Sensitivity to quinine is not uncommon.

OPERATIVE TREATMENT

It has been the experience of the majority of proctologists that the largest number of patients with hemorrhoids will be more satisfactorily relieved of their difficulty by the operative method of treatment.

The most acceptable type of operative treatment consists of excision and ligation. Clamp and cautery technics are largely being abandoned.

Details of operative treatment will not be outlined in this abstract. Suitable technics are described in any of the more recent proctologic texts, such as those by Buie, Yeomans, Bacon, and others.

In general, with all these technics which have minor variations, the following principles are employed: minimal attempts at preoperative sterilization of the area and the avoidance of strenuous preoperative catharsis and irrigations.

Caudal, spinal, or sacral anesthesia is the most satisfactory, if available. Many surgeons utilize anesthesia in oil preparations injected into the muscles at the time of operation for the control of postoperative pain. There may be abscess formation and other complications following this procedure and it is not recommended for the inexperienced.

The prone position is recommended for surgery, with its improved exposure and lessened bleeding over the lithotomy position. Undue trauma is avoided to the musculature and perianal tissues by unnecessary dilatations and retraction. When an actual narrowing of the anal canal due to fibrosis is present, division of the subcutaneous portion of the external sphincter muscle is indicated rather than an extensive dilatation.

Primary consideration is given at the time of operation to the removal of the primary piles. These are usually found in the right anterior, right posterior, and left central quadrants. Secondary piles may be excised as indicated. The mucosa of the anal canal is reconstructed to its normal position at the pectinate line when feasible. Adequate skin must be removed for the avoidance of skin tabs and an adequate open sulcus left for drainage.

POSTOPERATIVE CARE

Plugs and large packs are to be avoided. Snug, light vaseline dressings should be applied at the time of operation. If some oozing or uncontrolled bleeding is present, gelfoam or some other type of anticoagulant dressing is helpful.

Postoperative pain is controlled by the use of hot wet packs. Hot packs should be started on the anal area as anesthesia wears off. Renewal of hot wet dressings every hour for the first forty-eight hours will give adequate relief of postoperative discomfort. Sedation should be employed as necessary. The usual procedures may be necessary for difficulty in voiding.

After the first twenty-four or forty-eight hours, sitz baths may be adequate for cleanliness and the relief of discomfort. Three to four days of bed rest is probably all that is necessary for the average patient.

This postoperative period is an ideal time to correct existing bowel dysfunctions. A low residue or bland diet may be given from the beginning. If there have been severe symptoms of irritable colon, small amounts of tincture of belladonna and luminal with meals may be helpful. Excess roughage, mineral oil, and any other type of catharsis are to be avoided. Mineral oil tends to delay healing and predisposes to infection.

On the third postoperative day, a small oil retention enema may be given when necessary, followed by a one pint saline enema the following day. On the third or fourth day the anal canal should be gently dilated with a well lubricated gloved finger to prevent wound edges from falling together. These gentle dilatations should be continued every four or five days until the operative wound is completely healed. This will require a period of observation of three to four weeks. The postoperative use of ointments, greases, suppositories, and so forth, is to be condemned.

Consultation Service

This special consultation information service is offered as a regular monthly feature of *Postgraduate Medicine*. Readers are invited to call on this Service for answers to difficult medical problems from members of our Editorial Board best qualified to help. Each question will be answered by mail and those of general interest will be published each month. Address all communications to Consultation Service, *Postgraduate Medicine*, 516 Essex Building, Minneapolis 3, Minnesota.

ULCERATIVE COLITIS

QUESTION: What is the best treatment for ulcerative colitis, accompanied by anemia?

M.D.—Kentucky

ANSWER: Until the relative importance of the various factors concerned in the pathogenesis of chronic ulcerative colitis are better understood than they are at present, no "best treatment" applicable to all cases can be stated. Individualization in therapy empirically tried is the best that can be offered.

The most helpful procedures are as follows:

1. Complete rest in bed while there is fever and more than three stools daily.

2. High protein diet chiefly in the form of red meats, liver, kidneys, sweetbreads, and lean pork, with avoidance of coarse vegetables, fruits, and other high residue foods.

3. Cevitamic acid (vitamin C) 1 to 5 mg. daily depending on amount of fever, and vitamin B complex usually 2 capsules daily.

4. Iron and transfusions may be used for anemia, but response is not very satisfactory unless the underlying colitis is improved.

5. Most patients with ulcerative colitis have serious emotional problems and anxieties which need to be relieved, if possible; this is often difficult to accomplish.

6. No categorical statement concerning surgical intervention—ileostomy and possible subsequent colectomy—can be made, as resort to surgery is an individual case problem.

FRANCIS G. BLAKE

VITAMIN E FOR ANGINA PECTORIS

QUESTION: What has been the experience with the use of vitamin E in angina pectoris?

M.D.—Ohio

ANSWER: Although many animal experiments indicate biological activity of vitamin E, there are no well controlled observations to indicate that it has any effects in man. This may be due to the fact that the amounts of vitamin E in ordinary foods are adequate for human nutrition even on otherwise poor diets. The possibility exists that vitamin E may be synthesized in humans.

It should not be forgotten that angina pectoris is a subjective condition which may be influenced psychologically as well as by drugs.

Five cases of angina pectoris that had been followed in this clinic for several months were treated with daily doses—400 mgm. of mixed natural tocopherols for periods of one to two months. Control observations of exercise performance on a motor driven treadmill with continuous measurements of oxygen consumption, ventilation, heart rate and EKG, and blood pressure were obtained. Serial tests during treatment and while on placebo capsules showed no significant changes. There was no consistent subjective improvement while the patients received the drug.

WILLIAM S. MCCANN

TREATMENT OF BOILS

QUESTION: Is penicillin of value for the treatment of boils? Is x-ray treatment helpful? What measures may be taken to prevent their recurrence?

M.D.—Iowa

ANSWER: Penicillin is of marked value in the treatment of boils if given either systemically or locally. A daily injection of 300,000 units of penicillin in oil and beeswax for several injections usually causes the rapid healing of existing furuncles but does not prevent recurrences.

In single or relatively few lesions, infiltration of the furuncle with 50,000 units of penicillin per cubic centimeter of saline or distilled water usually causes rapid involution. A second injection of the boil after twenty-four hours may be necessary.

In chronic furunculosis extreme cleanliness of the body through daily multiple baths is a requisite. Autogenous vaccines, staphylococcus toxoid, and quinine given internally over a long period are of value. X-ray given empirically in the past, has been largely supplanted by use of antibiotics and sulfonamide drugs.

BEDFORD SHELMIRE

INDICATIONS FOR TONSILLECTOMY

QUESTION: Is tonsillectomy beneficial in cases of thyrotoxicosis, acute rheumatism, nephritis?

M.D.—California

ANSWER: Cases have been reported in which active thyrotoxicosis has disappeared following removal of infected tonsils.

For years it has been debated whether or not the removal of tonsils lessens the possibility of a child's developing acute rheumatic fever, and whether or not, following an attack of rheumatic fever, removal of the tonsils makes recurrences less likely.

In acute nephritis, it would seem wise to remove infected tonsils.

W. D. STROUD

NOTE: On page 78 of the January 1948 issue, part (e) of the answer to the question on pre-eclampsia diet should have read "Fat is *not* especially necessary. . . ."

New Drugs and Instruments

Information published in this department has been supplied by the manufacturers of the products described.

GLUCO-THRICIL

PURPOSE: An antibiotic nasal decongestant.

COMPOSITION:

Ephedrine (as the lactate) ..... 1 per cent
Tyrothricin 1:5,000

in an isotonic dextrose solution containing cetyl trimethyl ammonium acetate as a solubilizing and stabilizing agent.

INDICATIONS FOR USE: In relief of nasal congestion accompanying the common cold, acute catarrhal rhinitis, allergic rhinitis, acute rhinosinusitis, and acute ethmoiditis.

DOSAGE AND ADMINISTRATION: May be administered in the form of a spray, by drops, or nasal pack. Adult patients with acute, upper respiratory infections should be instructed to use Gluco-Thricil by spray or drops three or four times daily. Two to four drops should be instilled in each side of the nose. For children, one or two drops in each side of the nose three times daily will usually suffice.

HOW SUPPLIED: In 1-ounce bottles with dropper cap, and 1-pint bottles.

PRODUCER: Parke, Davis & Company, Detroit, Mich.

DIENESTROL "RARE"

PURPOSE: Synthetic, crystalline estrogen for oral administration.

INDICATIONS FOR USE: Menopausal therapy and treatment of suppression of lactation.

CAUTIONS: Contraindications of Dienestrol are similar to those for estrogens—both natural and synthetic, such as familial or personal history of malignancy of the reproductive system.

DOSAGE AND ADMINISTRATION: Menopausal syndrome: 0.1 to 0.5 mg. daily for mild to moderately severe symptoms; 0.5 to 1.5 mg. daily for severe symptoms or when climacteric has been artificially induced. Suppression of lactation: 0.5 mg. three times daily for the first three days; 0.5 mg. daily for the fourth through tenth day.

HOW SUPPLIED: In small, sugar-coated tablets in potencies of: 0.1 mg. (yellow), and 0.5 mg. (orange); bottles of 100 and 1,000.

PRODUCER: Rare Chemicals, Inc., Harrison, N. J.

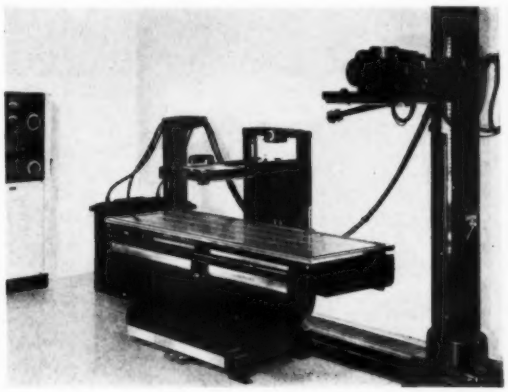
VIM LAMINEX NEEDLE

PURPOSE: Hypodermic needle.

DESCRIPTION: "Laminex" is the brand name for a new stainless steel for needle manufacture. It is a laminated stainless steel with the temper of high carbon steel.

All the former features of the VIM needle have been included in the manufacture of this new needle. VIM "Laminex" needles are available at regular VIM prices through all leading Surgical Supply Dealers. Free descriptive folder is available from:

PRODUCER: MacGregor Instrument Co., Needham 92, Mass.



MAXISCOPE 500

DESCRIPTION: High-voltage diagnostic x-ray apparatus. It mechanizes many operations, including the determination of exposure times, the automatic selection of spot-film areas, and automatic protection from excess radiation when the machine is adapted for flouroscopy. The machine has pedal-controlled, variable-speed hydraulic motor-drive for angulating the table and the automatic centering of exposure areas.

The voltage has been increased from 100,000 volts to 130,000 volts to provide clearer radiographs of dense portions. The x-ray table incorporates phosphorescent "eyes" which show all protruding parts for orientation of the physician, patient, and table during the darkness required for fluoroscopy.

PRODUCER: General Electric X-ray Corporation, Milwaukee 14, Wis.

PHENO-BEPADOL TABLETS

PURPOSE: Sedative and dietary supplement.

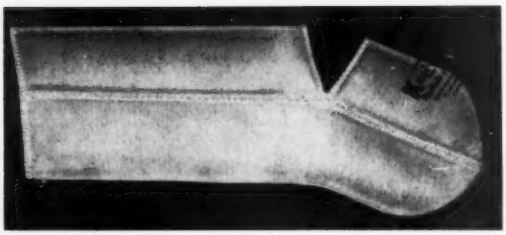
COMPOSITION: Each Pheno-Bepadol Tablet contains phenobarbital ¼ gr.; thiamine hydrochloride 3 mg.; riboflavin 2 mg.; niacinamide 10 mg.; calcium pantothenate 1 mg.; pyridoxine hydrochloride 0.5 mg.; secondary liver fraction and dried yeast.

INDICATIONS FOR USE: For patients suffering from neuroses, functional digestive disorders, nervous colitis, diarrhea, the nervous vomiting of pregnancy, overwrought nerves, hyperthyroidism, and simple insomnia.

DOSAGE AND ADMINISTRATION: One or two tablets, taken by mouth daily, as prescribed by the physician, and may be adjusted to the intensity of reaction desired for the individual patient.

HOW SUPPLIED: Bottles of 100 and 1,000 tablets.

PRODUCER: International Vitamin Division, Ives-Cameron Company, Inc., New York 16, N. Y.



KAST-SOCKS

DESCRIPTION: Surgical sock to protect the toes of persons wearing plaster leg casts, made of fine white combed cotton. Rubber binding inserted at both heel and top opening makes these socks fit neatly over the entire cast. They can be washed and used repeatedly.

PRODUCER: Plaut Products, Cincinnati 2, O.

VYCOM "B" TABLETS

PURPOSE: High-potency vitamin B complex for oral administration.

COMPOSITION: Each Vycom "B" Tablet contains:

Thiamine hydrochloride (vitamin B_1)	5 mg.
Riboflavin (vitamin B_2)	10 mg.
Nicotinic acid amide	150 mg.
Yeast (U.S.P.)	100 mgm.

INDICATIONS FOR USE: For nutritional supplementation in the vitamin B deficiency syndromes when oral therapy appears adequate, or during and after withdrawal of parenteral therapy. These indications include: beriberi, ariboflavinosis, and pellagra. Also useful pre- and postoperatively, and in pregnancy, lactation, fever, and hyperthyroidism, and in gastrointestinal disorders, where intake, absorption or utilization of the water-soluble vitamins is impaired.

DOSAGE AND ADMINISTRATION: One Vycom "B" Tablet daily, or as recommended in individual cases.

HOW SUPPLIED: In bottles of 100 tablets, sugar-coated red.

PRODUCER: Bristol Laboratories, Inc., Syracuse, N.Y.

DEKA

PURPOSE: Sedative expectorant.

COMPOSITION: Each fluid ounce contains codeine sulfate 64 mg. (1 gr.); calcium iodide 0.13 gm. (2 gr.); chloroform 0.13 (2 gr.), with wild cherry, tolu, squill, yerba santa, citric acid, and menthol. Alcohol content, 6.5 per cent, glycerin content, 3 per cent.

INDICATIONS FOR USE: In all cases of nonproductive cough other than tuberculosis.

DOSAGE: Adults, 1 or 2 teaspoonfuls; children, ½ or 1 teaspoonful. May be taken three or 4 times daily.

CAUTION: *It should not be used in tuberculosis!*

Idiosyncrasy to iodides, while not common, may be serious, and small doses may cause distressing symptoms of iodism in those who are sensitive.

HOW SUPPLIED: Bottles of 2, 4, and 16 fluid ounces and 1 U.S. gallon (128 fluid ounces).

PRODUCER: Winthrop Chemical Company, Inc., New York 13, N.Y.

ENDOGLOBIN-C WITH FOLIC ACID

PURPOSE: Treatment of anemic patients who require several individual medicinal agents for a mixed deficiency.

COMPOSITION: Each sugar-coated tablet (orange) contains:

Liver residue (secondary fraction, 50:1, derived from 10 gm. fresh liver)	0.2 gm.
Ferrous sulfate exsiccated	0.2 gm.
Thiamine hydrochloride	1 mg.
Riboflavin	0.66 mg.
Nicotinic acid	10.0 mg.
Ascorbic acid	50 mg.
Folic acid	1 mg.

DOSAGE AND ADMINISTRATION: Three to six tablets daily.

HOW SUPPLIED: Bottles of 40, 100, 500, and 1,000 tablets.

PRODUCER: Endo Products, Inc., Richmond Hill, N.Y.

PROGESTORAL TABLETS

PURPOSE: For high-potency oral luteal therapy.

COMPOSITION: Each tablet contains 25 mg. of pregneninolone (anhydro-hydroxy-progesterone).

INDICATIONS FOR USE: In the treatment of functional uterine bleeding and threatened abortion.

HOW SUPPLIED: Boxes of 20 and 100 tablets.

PRODUCER: Roche-Organon, Inc., Nutley, N.J.

ELUTEIN

PURPOSE: Protein supplementation of the diet.

COMPOSITION: Intact natural protein from nonagenized wheat flour (77 per cent), plus L-Lysine Hydrochloride (4 per cent). Total nitrogen: 13.4 per cent.

DESCRIPTION: A bland, palatable powder which may be taken plain or mixed into any number of food preparations. High dosages do not disturb the gastrointestinal tract.

DOSAGE AND ADMINISTRATION: 30 gm. of Elutein daily can maintain nitrogen balance in the average patient. Larger amounts will be indicated according to degree of nitrogen deficiency. Elutein may be incorporated into cakes, puddings, custards, cereals, etc., without creating any foreign taste.

HOW SUPPLIED: 1 lb. in a glass bottle, 12 bottles to a shipping carton.

PRODUCER: Interchemical Corporation, Biochemical Division, Union, N.J.

URESTRIN AND VITAMIN B

PURPOSE: Natural crystalline estrogens and vitamin B complex in a syrup base, for use in the treatment of combined hormonal and vitamin deficiencies.

COMPOSITION: Each fluidounce contains:

Estrogenic substances	30,000 I.U.*
Thiamine hydrochloride	30 mg.
Riboflavin	30 mg.
Nicotinamide	300 mg.
Alcohol	5 per cent

*Consisting of estrone 3 mg. and traces of other naturally occurring estrogens (estradiol, equilin, equilenin and hippulin).

DOSAGE AND ADMINISTRATION: One to three teaspoonfuls daily, depending on severity of symptoms.

HOW SUPPLIED: 12-ounce bottles.

PRODUCER: The Upjohn Company, Kalamazoo, Mich.

GLYTHEONATE

PURPOSE: Management of various related conditions of many cardiovascular patients such as hypertensive states accompanied by increased capillary fragility.

COMPOSITION: Each tablet (green-uncoated) contains:

Theophylline-sodium glycinate	325 mg. (5 gr.)
Phenobarbital	16.2 mg. (¼ gr.)
Rutin	20 mg.

CAUTIONS: May be habit forming.

DOSAGE AND ADMINISTRATION: 1 to 2 tablets three times a day or as determined by the physician. To be dispensed only by or on the prescription of a physician.

HOW SUPPLIED: Bottles of 100 and 500.

PRODUCER: The E. L. Patch Company, Boston, Mass.

MEPRANE-PHENOBARBITAL

PURPOSE: For relief of menopausal symptoms.

COMPOSITION: Each tablet contains:

Meprane (3,4-bis (m-methyl-p-propion-oxyphenyl) hexane)	1 mg.
Phenobarbital	0.16 mg.

CAUTIONS: Phenobarbital may be habit forming.

DOSAGE AND ADMINISTRATION: In menopause, initial therapy—3 Meprane-Phenobarbital tablets daily, in divided dosage. Physicians may replace part of this dosage with Meprane (without Phenobarbital) if less sedation is required. For maintenance therapy—Meprane-Phenobarbital, or Meprane, 1 or 2 tablets daily.

HOW SUPPLIED: Bottles of 100 and 500 tablets.

PRODUCER: Reed & Carnrick, Jersey City, N. J.

SODIUM SULFACETIMIDE NASAL SOLUTION 10%

PURPOSE: Relief of nasal congestion incident to the "common cold" and preventing complications.

COMPOSITION: Aqueous solution of sodium sulfacetimide with dl-desoxyephedrine hydrochloride 0.125 per cent buffered to pH 7.4.

DOSAGE AND ADMINISTRATION: Nasal instillation of 1 to 3 drops, four times daily, at the onset of the earliest symptoms of a cold is the preferred treatment.

PRODUCER: Schering Corporation, Bloomfield, N. J.

RESTROL

PURPOSE: Oral estrogenic therapy.

INDICATIONS FOR USE: Management of menopausal syndrome, treatment of suppression of lactation, amenorrhea, and other hypo-ovarian states.

CAUTIONS: Contraindications for Restrol are similar to those for natural estrogens, viz., familial or personal history of malignancy of reproductive organs.

DOSAGE AND ADMINISTRATION: Menopausal syndrome: 0.1 to 0.5 mg. daily for mild to moderately severe symptoms; one tablet of 0.5 mg. to three tablets of 0.5 mg. daily for severe symptoms. Suppression of lactation: Three tablets of 0.5 mg. daily, in divided doses for three days, followed by one tablet of 0.5 mg. daily for one week. Amenorrhea and other hypo-ovarian states: One or two tablets of 0.1 mg. daily, increased to 1.0 mg. daily as indicated.

HOW SUPPLIED: In small tablets of two strengths, 0.1 mg. (coated pink), and 0.5 mg. (coated green); bottles of 100, 500, and 1,000.

PRODUCER: Central Pharmacal Company, Seymour, Ind.

MINERALIGHT MODEL H

DESCRIPTION: Ultraviolet lamp, equipped with a Woods filter, and operating on 110 volt A.C. from standard electrical plug-in connections. A full guarantee is furnished by the producer. Model H Mineralight, in a light-weight aluminum and plastic case, is being made available as part of a kit containing fluorescent mineral samples. Full information can be obtained from:

PRODUCER: Ultra Violet Products, Inc., Los Angeles, Calif.

DELVINAL-ASPIRIN

PURPOSE: For relief of pain and nervous tension.

COMPOSITION: Each capsule contains 30 mg. (½ gr.) of Delvinal 5-ethyl-5-(1-methyl-1-butenyl) barbituric acid and 0.30 gm. (5 gr.) aspirin (acetylsalicylic acid).

INDICATIONS FOR USE: In myalgic, neuralgic and arthritic conditions, particularly in hypersensitive, neurasthenic or nervous patients, or those suffering from anxiety tension states. Useful in allaying fear and controlling pain preceding minor surgical operations.

DOSAGE AND ADMINISTRATION: One capsule of Delvinal-Aspirin vinbarbital and aspirin administered orally every three or four hours is recommended for the average adult dose. In pediatrics and geriatrics the dosage should be regulated accordingly. The capsules should be administered not more than one hour prior to performing any surgical or dental procedure.

HOW SUPPLIED: In bottles of 100 and 1,000.

PRODUCER: Sharp & Dohme, Inc., Philadelphia, Pa.

FLUOROSSTEOL

PURPOSE: For inhibition of dental caries.

COMPOSITION: Prepared from the hard, thigh bones (femur) of government inspected cattle. Approximately 95 per cent of the mineral matter present as a double salt of calcium carbonate and tribasic calcium phosphate, with an additional 1.5 per cent in the form of tribasic magnesium phosphate. Standardized to contain not more than 0.2 per cent of calcium fluoride.

INDICATIONS FOR USE: Of greatest value when administered to infants and children.

ADMINISTRATION: Orally. Infants and children under 3 years of age, 10 gr. daily in food or fluids; children between 4 and 8 years of age, 15 gr. daily, and children over 15 years of age or adults, three 5 gr. tablets daily.

CAUTION: Not indicated where the fluorine content of the water supply exceeds 0.5 parts per million. Fluorossteol is contraindicated in renal disease due to the danger of cumulative toxicity.

HOW SUPPLIED: In 5 gr. tablets and capsules.

PRODUCER: The Armour Laboratories, Chicago 9, Ill.

Leaves from a Doctor's Diary

By Maurice Chideckel

February 1 . . . They are still sentient men, those segments of mankind that have been stigmatized, repudiated, and incarcerated in the asylums for the insane. In many, tragedies shattered their brains and their lives; in others erratic parents made a black rent in the texture of their immature lives, and the rent widened as the years rushed on; a cruel stepfather or stepmother worked havoc with the autonomic nervous system and wrecked the highest functioning mechanism of living man, the mind; problems and conflicts, intrigues and disappointments, the rise and fall, the ebb and flow of stormy happenings, impeded, distracted, and eventually destroyed their mentalities; the blasting of cherished hopes; the ambitions, expectations, and certainties that were fused in the crucible of their imaginations and that proved but illusions, made wrecks of those who once were normal men and normal women. Then there were those whose lives developed against a background of confusion; hereditary taints, moral crimes, other causes and no causes at all.

I stood and faced them. There they are, living in a nonexisting world, divergent in mood and purpose, like the men and women living in a world of reality, sighing and weeping, laughing and dancing, planning and scheming, accumulating and spending imaginary millions; many with congeries of interest, others with no interest at all. The tiny spark of the normal was a monstrous blaze that they fought to extinguish, with defeat inevitable.

I always knew that the physical and emotional states are closely coordinated, the coordination being brought about by the endocrine glands and the autonomic nervous system. But I never knew and do not now, whether personality is determined by hereditary endowments, or by environmental conditions.

Here is John Thomas Audrey. His environments were ideal. His forebears for generations were sound physically and mentally. No insanities and no prodigies. From early childhood he had no problems, and no conflicts. What brought about his violent insanity? He certainly had no inability to meet social or economic demands. There were no somatogenic factors. What caused the elation, the overtalkativeness and overactivity? For days and nights at a time he quips and laughs and almost never stops talking. Mostly his speech is incoherent. As I stood facing him he was Emperor of Brazil. That Brazil is now a Republic was none of his business. I have seen powerful beasts of prey becoming exhausted in the struggle to escape. John Thomas Audrey never tires.

Next to him was the Emperor of Japan. Fred Tilden is his name. Once he was in that country and he evidently absorbed the mental climate of the East, for he bowed and swayed as do the Japanese. "Who," I asked, "informed your Majesty that you are the Emperor?" He assumed a serious mien as he answered: "Our Lord and Saviour, Jesus Christ told me so." From the next cell came a loud and angry voice: "He is a goddam liar. I never told him that."

Here is Mark Gallagher with depressive psychosis, with downcast face, refusing food and drink, weeping, wringing his hands, or sitting motionless. Not once did he raise his eyes when I attempted to talk to him. His attacks have periodicity. Between the attacks he is a perfectly well and pleasant man. In his sanity he told me of a middle-aged man who, he said, has suicidal tendencies. Why? Well, if I see his wife, I'll know the reason. But why did he marry her? She was earning fifty dollars a week. Now she lost her job.

And there were the syphilophobiacs, the claustrophobiacs, and the agraphobiacs. There were men with anxiety neuroses and some with compulsive ideas. A Mrs. Goodwin never stopped washing her hands, because someone touched her hands, and there was the obsession of contamination. The schizophrenics and the paranoiacs, and the alcoholic psychotics. Those with hallucinations of sight and hearing and smell. And those with disorders of idea associations. Withal it is a terrible world that I was in. Incarcerate a normal man among those abnormals, and he too would become mentally unbalanced. I must have looked depressed. A visitor called to the guards, pointing at